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DISSERTATION

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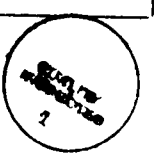
The Graduate School

University of Kentucky

1988



EFFECT OF VARIATION IN THE BURST MODE AND CARRIER FREQUENCY OF HIGH INTENSITY ELECTRICAL STIMULATION ON MUSCLE FATIGUE AND PAIN PERCEPTION OF HEALTHY SUBJECTS

DISSERTATION

A dissertation submitted in partial fulfillment of the requirements for the degree of Doctor of Education at the University of Kentucky

By

James Grattan Rooney

Lexington, Kentucky

Director: Dean P. Currier, Professor of Physical Therapy

Lexington, Kentucky

1988

(a) Electrical stimulation of human skeletal muscle using these conditions results in a decrease in torque and a decrease in the area under the negative deflection of the EMG signal recorded from the stimulated muscle. The degree of torque loss and of EMG area decrease is dependent upon the frequency and amplitude of the stimulation; (b) The amplitude of stimulation (mA) necessary to bring about 50% MVC of a given muscle group is dependent upon the frequency of the stimulating current. All of the frequency combinations used in this study were capable of stimulating the quadriceps femoris muscle to 50% MVC; (c) Subjects pain tolerance is contingent upon the stimulating frequency and the amplitude of stimulation. Variations in either burst mode or carrier frequency of a stimulating current can alter a subject's pain response; (d) When torque production decreases as a result of electrical stimulation, an increase in current amplitude will re-establish the desired torque level (50% MVC); (e) In the context of this study, there was no correlation between torque and EMG changes following electrically induced muscle stimulations; (f) In addition to the discomfort encountered during electrical stimulation of the quadriceps femoris muscle to 50% MVC, there may be delayed onset of muscle soreness occurring 24 to 48 hours after stimulation.

James Grattan Rooney

James Grattan Rooney

17 October 1977

Date

ABSTRACT OF DISSERTATION

EFFECT OF VARIATION IN THE BURST MODE AND CARRIER FREQUENCY OF HIGH INTENSITY ELECTRICAL STIMULATION ON MUSCLE FATIGUE AND PAIN PERCEPTION OF HEALTHY SUBJECTS

This study determined the effects that electrically stimulating human skeletal muscle at various combinations of burst modes and carrier frequencies have on muscle fatigue and pain perception. It was carried out on 27 healthy subjects. An Electrostim 180 2i that was capable of delivering nine combinations of burst mode and carrier frequency was used as the stimulating unit. Subjects were stimulated at a current amplitude sufficient to produce 50% of their maximum voluntary contraction (MVC) and received a series of ten electrically induced contractions at each frequency combination. A 15-second stimulation followed by 50 seconds of rest was the duty cycle utilized. The Cybex II dynamometer was used to record the torque data and the Cadwell 5200A registered the EMG signals. The results of the data analysis allow the following conclusions to be drawn:

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TABLE OF CONTENTS

Page

Page

LIST OF TABLES

LIST OF FIGURES

Chapter 1

INTRODUCTION AND THE PROBLEM

Claims of using electrical stimulators to develop muscle strength (torque, force), improve athletic performance, and increase muscle size have intensified interest in this modality as a rehabilitative and performance enhancing tool (Currier & Mann, 1983; Gould, Donnermeyer, Gammon, Pope, Ashikaga, 1983; McMiken, Todd-Smith & Thompson, 1983; Halback & Straus, 1980; Johnson, Thurston & Ashcraft, 1977; Kots, 1975; Kots, 1976; Kramer & Mendryk, 1982; Kramer, 1987; Laughman, Yodas, Garrett & Chao, 1983; Morrissey, Brewster, Shields & Brown, 1985; Nitz & Dobner, 1987; Owens & Malone, 1983; Peckham, Mortimer, Mursolais, 1975; Romero, Sanford, Schroederm & Fahey, 1982; Selkowitz, 1982; Stanish, Valiant, Bonen & Belcastro, 1982). Kots' claims in 1975 and 1976 of 100% increases in strength of untrained healthy subjects and 40% increases in strength of trained athletes served as the impetus to these investigations (Kramer & Mendryk, 1982). Kots stated that in order for electrical stimulation (ES) to be effective, two criteria must be met. First, the current must be one of adequate magnitude and frequency to produce strong tetany in the muscle, and secondly, the stimulus must be relatively tolerable. Many researchers have shown that ES without voluntary effort can increase muscle strength (Currier & Mann, 1983; Halbach & Straus, 1980; Kots, 1977; Kramer & Semple, 1983; Laughman, Yodas, Garrett & Chao, 1983; Liu, 1984; McMiken, Todd-Smith & Thompson, 1983; Nobbs, Rhodes, McKenzie, Taunton & Mattison, 1982; Owens & Malone, 1983; Romero, Sanford, Schroederm & Fahey, 1982; and Selkowitz, 1982). However, these researchers report that patient discomfort is a major

drawback to this treatment technique. Kots' reports of stimulating subjects to 120% of their maximum voluntary contraction (MVC) level without pain have not been substantiated by these other studies.

Kots noted that by using a 2,500 Hz carrier frequency modulated at 50 bursts per second, an adequate pain free training effect could be elicited. Kots attributes the pain free characteristic of this combination of frequencies to the currents blocking the pain input of small afferent nerve fibers by activation of the larger efferent motor fibers (Balogun, 1987). Vodovnik, et al. (1965) and Crochetier, et al. (1967) reported decreased skin sensitivity to ES while using frequencies above 500 Hz. Frequencies above these levels apparently do not initiate a pain response from sensory fibers (Li & Bak, 1976). Therefore, frequencies of greater than 500 Hz seem to satisfy Kots' second criterion for an effective stimulus—i.e., a relatively tolerable stimulation. It may be that the 2,500 Hz carrier frequency advocated by Kots produces an anaesthetic effect. However, frequencies of greater than 500 Hz do not produce optimum muscle torque. Stimulating frequencies of greater than 500 Hz may be stimulating a muscle during its absolute refractory period (ARP) and be too rapid to fully activate the muscle for optimal torque production (Massey, Nelson & Sharkey, 1965). Moreno-Aranda & Seireg (1981) reported that the ARP for skeletal muscle was approximately 4 to 5 ms. This reduced torque response means that for optimum performance a muscle should be allowed to rest at least 5 ms. between contractions. These authors reported that maximum muscle force of the quadriceps femoris muscle is produced at frequencies between 70 and 150 Hz. Edwards, Young, Hosking & Jones (1977) reported that the frequencies of electrical stimulators used to produce tetanic muscular contractions

range from 33 to 1,000 pulses per second. The reported studies using this wide range of frequencies, coupled with variations in wave forms and current amplitudes have made comparing and evaluating them very difficult. The present study will provide information on the effect that various combinations of burst frequency and carrier frequency have on pain perception and muscle force (torque). A carrier frequency is a continuous fast rate of pulses. When this carrier frequency is interrupted (modulated) to provide groups of these fast rate of pulses, the group or package of pulses is then called a burst.

Fatigue is another aspect of muscle performance that will be assessed during this research. Exercise physiologists report that the stimulus for muscle growth is the tension developed within the muscle (Astrand & Rodahl, 1977; Edington & Edgerton, 1976; Goldberg, Etburger, Goldspink & Jablecki, 1975; and McArdle, Katch & Katch, 1986). The greater the muscle tension developed during each contraction, the greater the potential for growth. If a muscle could be stimulated maximally during each contraction of an exercise bout without fatiguing, maximal torque could be developed with each repetition. These maximal contractions would then potentiate optimal strength gains. Kots stimulated subjects to 200% of their MVC and reported minimal fatigue occurring during 10 contractions (Kramer & Mendryk, 1982). However, Currier & Mann (1983), using electrical stimulation said to duplicate the characteristics described by Kots and stimulating sufficiently to produce the equivalent of 60% of a subject's MVC, reported significant muscle fatigue (torque decrement/electrically induced contraction) during a bout of 10 electrically stimulated contractions. Selkowitz (1985) and Moreno-Aranda & Seireg (1981) also showed fatigue while stimulating

muscles at various burst frequencies and carrier frequencies. If Kots' claims of minimal fatigue during maximal electrical stimulation could be duplicated clinically, an ideal training vehicle would exist. Maximal pain free muscle contractions with minimal fatigue would allow optimal activation of all the responding motor units during each repetition of an exercise bout. Selkowitz (1985) has shown that there is a direct relationship between the intensity of stimulation and strength gains during the ES of normally innervated muscle.

Gollnick (1973a,; 1974a, b) found that muscle contractions of less than 20% of a muscle's MVC utilized slow twitch muscle fibers, while tensions exceeding 20% of the MVC called for the addition of fast twitch fibers. Goldnick's work supports the "size principle' espoused by Henneman (1965) and confirmed by electromyographic studies using fine wire electrodes (Hannez, 1974) and bipolar surface electrodes (Gydikow & Kosarov, 1974). There have also been reports of initial activation of fast twitch motor units during voluntary ballistic activity (Grimby & Hannerz, 1977). Appell reported that during high intensity ES, there is a reversal of the "size principle" for volitional contractions in that the largest motor neurons innervating the Type II fibers are activated first and to a greater degree than Type I fibers (Appell, 1988). Appell speculates that the selective activation of Type II fibers during high intensity ES may lead to hypertrophy of these fibers. However, the preferential activation of the fast-fatiguing Type II fibers by ES can lead to an increased rate of muscle fatigue (Currier & Mann, 1983; Peckman-Barun, 1988). This increased rate of fatigue can have deleterious consequences during functional electrical stimulation (FES).

This research project will compare the levels of muscle fatigue produced by varying combinations of burst frequency and carrier frequency during an exercise bout and speculate as to the type of motor unit being activated by the electrical stimulus. The rate of muscle fatigue will be analyzed and compared with changes in the pre- and post-stimulation electromyographic signals. I hypothesize that muscle fatigue (torque decrement) and pattern of EMG changes revealed in the raw EMG signals (pre- vs post-stimulation) are indicative of the early fatigue of the Type II muscle fibers. This early fatigue of the Type II units may be one of the reasons for the shift of Type II muscle characteristics to Type I characteristics during electrical stimulation (Eisenberg & Salmons, 1981; Eriksson & Haggmark, 1979; Greathouse, 1986 and Heilmann & Pette, 1979). In athletes performing high intensity ballistic movements and in the geriatric population--reported to have selected Type II fiber atrophy (Anlanssen, 1986)--the application of ES to prevent atrophy may be counterproductive considering Type II fibers take on Type I characteristics. However, the effect that electrical stimulation has on a particular fiber type may be related to the frequency and amplitude of the stimulator (Cabric, Appell & Risic, 1988). Cabric, et al. reported changes in the nuclear configuration of both the Type I and II muscle fibers following ES but showed more pronounced changes in the Type II fibers. In fact, using a sinusodal wave form with a 2500 Hz frequency and an intensity of 35 to 45 mA, Cabric reported Type II fiber hypertrophy. Minimizing Type II fiber fatigue should allow maximal muscle activation, maximize the desired adaptive strength changes, and minimize the rate of muscle fatigue.

The site of muscle fatigue has been debated for the last 60 years (Bigland-Ritchie, Jones, Hosking & Edwards, 1978; Bigland-Ritchie & Woods, 1984; Merton, 1954; and Reid, 1928). The areas cited in the literature are areas that lie within the central nervous system (central fatigue), the metabolic and enzymatic processes within the muscle fiber (peripheral fatigue), and the excitation-contraction coupling process that tie the central and peripheral elements together. Although biochemical analysis of the fatigued muscle would be necessary to objectively identify metabolic changes in the muscle, this author will speculate on the cause and location of electrically induced muscle fatigue.

In the laboratory during controlled experiments on animals and in limited clinical trials on humans, researchers have been able to prevent muscle atrophy and in some cases, produce muscle hypertrophy (Cabric, et al., 1988; Greathouse, 1985; Kots, 1976; Munsat, McNeal & Waters, 1976; and Nitz & Dobner, 1987) by using electrical stimulation. High intensity ES with pulsed tetanic frequencies are capable of producing the level of muscle activity necessary to produce adaptive changes in the muscle. However, these same ES stimulus characteristics (frequencies and intensity) cause levels of fatigue and discomfort that limit the general clinical use of ES. Therefore, an optimal frequency combination that reduces pain and fatigue and yet maintains acceptable torque levels is needed to make ES a clinically accepted procedure.

The Problem

Statement of the Problem

The lack of knowledge of electrical stimulation as a therapeutic instrument has limited the utilization of this modality as a rehabilitative tool. What are the optimal parameters of electrical stimulation (amplitude, carrier frequency, pulsed bursts) that minimize subject fatigue (torque decrement) and yet are capable of stressing muscle to 50% MVC? Defining these parameters would enable physical therapists to design clinical programs that minimize muscle fatigue, prevent muscle atrophy and modulate pain perception.

The purpose of the present study was fivefold. The first objective was to determine how the amount of electrically induced muscle fatigue varied with different combinations of carrier frequency and pulsed bursts. The second intent was to determine the magnitude of current (mA) necessary to produce 50% of a subject's MVC at varying combinations of carrier frequency and bursts. The third goal was to examine a subject's response to the various combinations of carrier frequency and bursts to determine which is the least painful. A fourth objective was to determine what effect periodically increasing the stimulus frequency has on fatigue and torque production. The final aspect of this study was to compare raw EMG signals in the pre- and post-exercise periods to the amount of strength (torque) decrement. Although no biochemical analysis was performed, the author speculated on the site and cause of electrically induced muscle fatigue. The null hypothesis was that variations of carrier frequency and bursts have no effect on the amount of muscular fatigue or the subject's pain perception, and that there is no correlation between the raw EMG signals and the amount of muscle

fatigue. The experimental hypothesis is that there is an optimal combination of carrier frequency and bursts that sufficiently stresses muscle to 50% MVC while minimizing fatigue and perceived pain.

Scope of the Study

This study was carried out in two phases on 28 healthy volunteers between the ages of 18 and 35 years. One subject was unable to tolerate the necessary levels of stimulation and was dropped from the study. The majority of the subjects were recruited from physical therapy and physical education students at the University of Kentucky. Phase I determined the combination(s) of carrier frequency and bursts during ES that satisfied the following criteria:

1. Produces a minimum of 50% of each subject's MVC, and
2. Produces the least amount of discomfort to subjects being stimulated to 50% of their MVC.

These combined conditions are considered the optimum for clinical electrical stimulation.

Phase II, using frequency information obtained in Phase I, determined the effect of manually incrementing the current intensity of this optimum frequency after every series of two muscle contractions by ES has on muscle fatigue and torque production.

Phase I began with a familiarization session. During this familiarization session, the subjects' height, weight, sex, and age were recorded. The subject's maximal isometric knee extensor torque (the highest torque reading of three repetitions was used as the maximum) was determined using the Cybex isokinetic exercise unit, 50% of this maximal was used as a target torque during electrical stimulation. Following the torque measurement(s), each subject was given sufficient time to familiarize himself with the Electrostim 180-2i high intensity electrical

stimulation unit. The Electrostim 180-2i uses surface electrodes. Subjects were encouraged to stimulate their quadriceps femoris muscles with a current amplitude sufficient to produce 50% of their measured maximal knee extensor torque. During the familiarization session, individual subjects may produce greater or less than 50% of their maximal torque depending on individual tolerance to electrical stimulation. Fifty percent of the maximal torque was a target figure. Following the familiarization session, there were three additional experimental treatment sessions during Phase I. During each of these three experimental sessions, each subject randomly received one of three predetermined burst modes (50; 70; 90 Hz) combined with a randomly chosen carrier frequency (2,500, 5,000, 10,000 Hz). In a single test session, each subject received a series of ten stimulations at an intensity sufficient to produce approximately 50% of their maximal voluntary torque to determine the maximal amount of muscle fatigue. Fifteen seconds of stimulation was followed by a 50-second rest period. Following a five-minute rest period, each subject received another randomized combination of bursts and carrier frequencies and the frequency-torque-fatigue relationship was again determined by assessing the recorded torque curves. During the course of three treatment sessions, each subject experienced all combinations of the predetermined frequencies (total = 9). Immediately following the tenth stimulation (muscle contraction), the subject rated the pain perception using a 10 cm visual analog scale. A raw EMG signal was recorded during a maximal voluntary isometric contraction both pre- and post-stimulation. Adequate time for warm-up was afforded each subject prior to each experimental session and consisted of 3 to 5 minutes of stretching the leg muscles

supervised by a physical therapist. The hamstring, quadriceps femoris and gastrocnemius-soleus muscle groups were stretched (muscle elongation) using static stretching techniques. Subjects were positioned to stretch these muscle groups by the researcher, told to stretch to the point of tightness and hold for 15 seconds. A minimum of five stretches were performed for each muscle group. Following Phase I, torque/frequency graphs were analyzed to determine the optimal frequency (or frequencies) that produce 50% of a subject's MVC and are tolerable. A two-way ANOVA with repeated measures (frequency and carrier frequency) was used to analyze the data. The interaction of carrier frequency and pulsed bursts on pain, torque, and fatigue was analyzed using a Statistical Analysis System (SAS) statistical package. The SAS was also used to analyze the relationship between fatigue and any quantifiable changes in the EMG signal as well as the relationship that the burst and carrier frequency combination and the number of mA necessary to produce 50% of each subject's MVC.

Phase II determined what effect incrementing the intensity of the optimal frequencies has on fatigue and torque production. The Phase I optimal combination of frequency and carrier frequency was applied to each subject. After every two contractions, the intensity of the stimulation was increased to subject tolerance in order to maintain torque levels of 50% MVC to determine the effect this approach has on fatigue. Each subject received ten stimulations at the chosen frequency and experienced only the frequency combination they found to be most comfortable during Phase I.

As in Phase I, the SAS statistical package was used to analyze Phase II data. A significance level of .05 was used in the analysis of both

phases. A correlation between subjects weight and maximal voluntary isometric torque was also utilized.

Limitations of the Study

This study was designed to determine the combination of burst frequency and carrier frequency that could produce 50% of a subject's MVC and at the same time be tolerable to the subject. Although all the subjects tested found some combinations to be more comfortable than others, no subject reported any combination--applied at sufficient current amplitude to produce 50% of their MVC--to be comfortable. Several subjects complained of delayed muscle soreness occurring 24 to 48 hours after the electrical stimulation. This limitation could not be controlled. With encouragement and coaching, all of the subjects, but one, were able to tolerate the nine frequency combinations.

The use of muscle biopsies to the quadriceps femoris muscle following voluntary and electrically induced muscle contractions would have allowed direct observation of the metabolic changes following exercise.

Chapter 2

REVIEW OF LITERATURE

This chapter presents a review of literature that focuses on the clinical problems encountered when electrical stimulation is used to prevent muscle atrophy and strength loss. Muscle fatigue and the failure to generate adequate muscle torque, coupled with a patient's inability to tolerate the discomfort encountered during stimulation are the factors most often cited as limiting the clinical use of this modality (Halbach and Straus, 1980). Although there is little doubt that electrical stimulation will alter the subcellular and metabolic properties of the muscle (Cabric, et al., 1988; Ericksson & Haggmark, 1981; Greathouse, Nitz, Matulionis, & Currier, 1986; and Poorman & Taylor, 1979), the dual dilemma of fatigue and pain limit its use in the clinic. However, researchers have shown that by varying the frequency of the electrical stimulus, a subject's metabolic and pain perceptual response can be altered (Crocnetiere, Vodovnki & Reswick, 1967; Edwards, 1973; Vodovnik, Long, Regenos & Lippay, 1965). An optimal frequency or combination of bursts and carrier frequency that reduces pain and fatigue and yet maintains acceptable torque levels is needed to make electrical stimulation a viable clinical tool.

Electrical Stimulation and Muscle Fatigue

The site of muscular fatigue has been debated for the last 60 years (Bigland-Ritchie, Jones, Hosking, & Edwards, 1978; Bigland-Ritchie, 1981; Bigland-Ritchie & Woods, 1984; Bigland-Ritchie, Furbush & Woods, 1986; Edwards, 1975, 1984; Gibson & Edwards, 1985; Merton, 1954; Reid, 1928). The areas cited in the literature are those that lie within the central

nervous system and cause "central fatigue", the metabolic and enzymatic processes within the muscle fiber "peripheral fatigue", and the excitation-contraction coupling process that ties the central and peripheral elements together. During a voluntary muscle contraction, there is a chain of events starting with the motor cortex, traversing the motor tracts to the axon, crossing the myoneural junction, and finally terminating with the energy dependent interaction of the actin and myosin filaments (Bigland-Ritchie, 1981). Admittedly this is a simplistic view of the chain of motor activity and it must be remembered that there are many feedback loops in the chain that grade and guide muscle performance (Astrand & Rodahl, 1977; Schmidt, 1983). Edwards (1981) defined fatigue as the, "inability of a muscle or a group of muscles to sustain the required or expected force." Any interruption along this pathway could result in the decrease of muscle force that marks fatigue. Many researchers view fatigue on a continuum and believe that fatigue results from a series of interrelated events occurring at several points on the motor chain. These interruptions in the chain, be they chemical or mechanical, result in a loss of force. In addition, the site or combination of sites that fail initially during exercise may be due to the type and intensity of the exercise (Bigland-Ritchie, 1981).

The concept of central fatigue is supported by Asmussen (1979) and Ikai (from Bigland-Ritchie, 1984). These authors reported that a muscle exhibited a greater rate of force loss during a voluntary contraction than recorded during electrical stimulation. The authors theorized that a loss of drive from the motor cortex was responsible for the loss of force during the voluntary effort, hence "central fatigue". The research of Asmussen (1979) and Ikai (1961) supports the earlier work of Musso

(1915), Reid (1928) and Lippold (1960). Asmussen and Magin (1978) show that more work could be done following an exhaustive bout of exercise if subjects engaged in diverginal physical or mental activities during the rest period. Here the authors conclude that the balance between the facilatory and inhibitory impulses in the CNS is changed and consequently reduces central fatigue. Before 1954, only a fraction of the available motor units were considered to be activated during maximal voluntary muscle contractions. This misconception made the concept of central fatigue difficult to study, since all the motor units in a muscle could not be simultaneously activated by a voluntary effort. In that year, both Merton (1954) and Bigland & Lippold (1954) showed that in well motivated subjects, the MVC of the adductor pollicis muscle could match the force generated by the muscle during a supramaximal stimulation of the ulnar nerve. These studies were duplicated by Belanger and McComas (1981) and by Bigland-Ritchie, et al. (1978) using different muscle groups. These findings led these and other researchers to conclude that either MVC or the supramaximal stimulation of the nerve innervating a muscle could result in the complete activation of the muscle (Bigland & Lippold, 1954; Merton, 1954; Edwards, 1975; Bigland-Ritchie, Jones, Hosking & Edwards, 1978). These investigators speculate that during maximal muscle contractions lasting up to 60 seconds, central factors were not responsible for decrements in muscle force. In fact, Merton, Hill & Merton (1981) later reported that the force lost during a sustained MVC could not be restored with direct stimulation of the motor cortex or by stimulating the muscle membrane itself. In well motivated healthy subjects, there is little evidence supporting the role of central fatigue in the force loss noted during exercise that lasts 60 seconds or

less (Bigland-Ritchie, Johansson, Lippold & Woods, 1983; Bigland-Ritchie & Woods, 1984; Merton, 1954). During a sustained MVC (lasting more than 60 seconds) Bigland-Ritchie, et al. (1978) showed that central drive tends to decline over time and central fatigue may be due to a decrease in subject motivation or a decrease in motor neuron excitability. Pandolf (1978) suggests that subject motivation is decreased by pain and discomfort in the muscle during prolonged (greater than 60 seconds) muscle contractions. The author speculates that the buildup of lactic acid and the subsequent decrease in muscle pH stimulate free nerve endings in the muscle causing pain and leading to decreased motivation (Pandolf, 1978). Karlsson, Funderburk, Essen & Lind (1975) also cite pain from lactic acid accumulation as a limiting factor in exercise which may lead to decreased central drive. In a recent study by Bigland-Ritchie, et al. (1986), the authors stated that during intermittent submaximal voluntary contractions there is no evidence of central fatigue in the quadriceps femoris or the adductor pollicis muscles. This study confirmed the earlier work of Bigland-Ritchie, et al. (1983) that reported no evidence of central fatigue during intermittent submaximal limb exercise lasting up to 20 minutes. Both of these studies conflict with Bellemare's, et al. (1983) conclusion that decrease in central drive is a factor in fatigue.

The argument for fatigue occurring at the myoneural junction during submaximal voluntary muscle activity receives little support from the current literature (Bigland-Ritchie, Furbush & Woods, 1986; Bigland-Ritchie, Kukulka, Lippold & Woods, 1983; Marsden, Meadows & Merton, 1983; and Merton, Hill & Morton, 1981). Merton's work showing that force lost during MVC could not be restored by direct stimulation of

the muscle fibers offers convincing evidence that fatigue is not taking place at the myoneural junction during voluntary muscle activity. Bigland-Ritchie, et al. (1982) confirm Merton's conclusions as does the work of Hill, McDonnel & Merton (1980). These authors conclude that the main area of muscle fatigue during a voluntary contraction is in the contractile apparatus and not the myoneural junction. There is some evidence, however, that during electrical stimulation at 80 Hz, fatigue may occur because of reduced excitability of the muscle fiber or a decreased responsiveness at the myoneural junction (Jones, Bigland-Ritchie & Edwards, 1979). Elmqvist and Quastel (1965) reported increased release of neurotransmitter substances at the myoneural junction as the frequency of electrical stimulation increased from 30 to 70 Hz. The rate of fatigue at these frequencies during voluntary contractions of healthy muscle is not as great because of the natural progressive slowing of motor unit firing rates during maximal voluntary efforts. Jones, et al. (1979) reported that by changing from higher to lower (80 Hz to 20 Hz) frequencies of electrical stimulation, the force of a muscle contraction could be maintained and fatigue delayed while utilizing electrical stimulation. The force of an electrically stimulated muscle contraction may depend upon the stimulation frequency (Edwards, 1978). During a MVC of the adductor pollicis muscle the initial motor unit firing rate is 100 impulses per second and progressively slows to 20 per second in less than one minute. This decrease in motor unit activation rate enables the muscle to maintain its external force development and reduces the rate of fatigue (Marsdon, et al., 1983). Obviously, this rate of change is not seen with electrically stimulated contractions because of the constant stimulating frequency

(synchronous recruitment). The exact mechanism for the slowing of the motor unit firing rate is not known, but Marsdon, et al. (1983) speculate that it may be reflexly controlled with the cerebellum setting the pattern of muscle activation. Bigland-Ritchie & Woods (1984) also speculate that reflex feedback from the muscle is necessary to control the frequency of activation and consequently, the type and number of motor units activated. This frequency control enables the muscle to generate the minimal force necessary for a desired outcome. These researchers (Marsden, et al., 1983; Bigland-Ritchie & Woods, 1984) further speculate that this rate control during a voluntary contraction prevents fatigue at the myoneural junction. This may not be the case when electrical stimulation is utilized.

A recent study by Bigland-Ritchie, et al. (1986) offers further evidence that as the internal environment of a muscle changes during the contractile process, the rate of neuromuscular firing is controlled by a peripheral reflex within the muscle fibers. These authors monitored the motoneuron discharge rate during the first 10 seconds of a MVC. The contraction lasted an additional 10 seconds and then three minutes of rest was allowed the subject. After the rest period, the motoneuron discharge rate was again recorded during a MVC and the rate was found to be within 95 $\pm$/10% of the initial rate. The same protocol was followed on another group of subjects except their circulation was occluded in the tested extremity during the rest period. With the blood supply occluded, the motoneuron discharge rate during the second contraction remained depressed (Bigland-Ritchie, Dawson, Johansson & Lippold, 1986). The authors submit this as additional evidence that a peripheral mechanism within the muscle controls the firing rate of the motoneuron during a

contraction. This rationale may account for the different rates of fatigue during voluntary and electrically stimulated muscle activity. Currier & Mann (1983) reported a 24 percent decrement in the amplitude of fatigue curves of the quadriceps femoris muscle during ten repetitions of electrical stimulation (2,500 Hz carrier frequency modulated at 50 bursts per second) compared with a 10 percent decrement in subjects performing voluntary isometric exercise at a similar force. Hoskins, Young, Dubowitz & Edwards (1978) stated that force decrement during electrical stimulation was dependent upon the frequency of electrical stimulation. When stimulating the quadriceps femoris muscle at 30 Hz, a 10 percent decrease in force was reported. With a stimulating frequency of 100 Hz, a 59.6 percent force (decrement) was noted. This characteristic loss of muscle force occurred whether the muscle was stimulated directly as in the Hoskin and Young study or if the femoral nerve was stimulated indirectly (from Nelson & Currier, 1987).

Peripheral fatigue results from failure of the muscle contractile apparatus and yields a decreased force generating capacity in the individual muscle fibers (Bigland-Ritchie, Furbush & Woods, 1986). Changes in the contractile apparatus may be due to a decrease in energy supply or alterations in the contractile mechanism (Bigland-Ritchie, et al., 1986; Edwards, 1975; Gibson & Edwards, 1985; Hultman, Spriet & Soderlund, 1986). The immediate source of energy in the muscle is the breakdown of adenosine triphosphate (ATP) (Astrand & Rodahl, 1977). ATP can be replenished in the muscle by phosphocreatine (PCr) degradation, anaerobic glycolysis and oxidative phosphorylation of carbohydrate and fat. The manner in which ATP is replenished depends upon the type of muscular activity. At rest, free fatty acids constitute the main fuel

supply; during submaximal exercise, glycogen is utilized via oxidative phosphorylation; and during maximal effort, phosphagens and muscle glycogen are utilized anaerobically (Astrand, 1977; Hultman, Spriet & Soderlund, 1986). During different phases of exercise, one fuel source may dominate but the muscle does not utilize one fuel exclusively during any phase of exercise. The chemical changes noted during submaximal exercise can contribute to the loss of force seen with fatigue. The breakdown of PCr and glycogen during exercise increase the concentration of lactic acid (LA) and hydrogen ions (H+). These substances diffuse through the surrounding interstitial tissue. The more intense the muscle contraction, the more H+ ions are produced and the lower the muscle's pH becomes. Ericksson, Haggmark, Kressling & Karlsson (1981) report that the metabolite changes observed during intense muscular work (decreased PCr, decreased ATP and decreased glycogen stores) are also seen during electrically stimulated muscle contractions. The amount of LA and subsequent H+ ions produced may depend upon the predominent fiber type of the exercising muscle.

Komi and Tesch (1979) report that muscles with predominant Type II fiber compositions not only produce high torque values, but also have a greater susceptibility to fatigue than muscles with predominant Type I fibers. Predominance of fiber type may relate to Karlsson's observation that with Type II dominant muscles, more lactic acid is produced at a given exercise intensity (Karlsson, Sjodin, Jacobs & Kaiser, 1981). Komi and Tesch (1979) cite increased rates of fatigue and increased lactate formation in muscles rich in Type II fibers. Fitts and Holloszy (1976) state that decreased muscle pH might be responsible for contraction failure. As the concentration of Type II fibers increases, the amount of

LA produced increases (Stainsby & Eitzman, 1986; Tesch & Karlsson, 1977; Tesch, Sjoden, Thorstensson & Karlsson, 1978) Shalin, Edstrom and Sjoholm (1983) suggest that the decrease in intracellular pH is associated with muscle fatigue. Hermansen (1981) demonstrated that the pH of skeletal muscle decreases from about 7.0 at rest to 6.4 at exhaustion and speculates that this change in pH is responsible for the decreased muscle force noted at exhaustion. The author cites previous studies by Danforth (1965) and Ui (1966) that the two key enzymes promoting glycolysis -- phosphorylase and phosphofructokinase -- are almost completely inhibited at a pH of 6.4. With glycolysis inhibited, ATP formation is limited and force production capability decreases. The increase in H+ ion concentration may also limit the interaction of calcium and troponin necessary to combine actin and myosin in the contractile process (Astrand & Rodahl, 1977; Basmajian & DeLuca, 1985; Hermansen, 1981; Robertson & Kerrick, 1979; and Tesch, 1980). All of the adverse chemical reactions associated with the decrease in pH may contribute to peripheral fatigue. In addition, the decrease in pH may cause a reduction in the conduction velocity of the muscle membrane and myoelectric shift toward the lower neuromuscular firing rate (Basmajian & DeLuca, 1985; Hagberg, 1981; Petrofsky, 1979; Petrofsky & Lind, 1980). Several authors have tied this change in muscular activity, which can be quantified using surface EMG (Fowler, Danta & Gilliatt, 1971) to the decrease in the conduction velocity of the sarcolemma caused by the increase in lactic acid concentration seen during muscle fatigue (Bigland-Ritchie & Woods, 1984; Broman, 1977; Broman & DeLuca, 1985; Komi & Tesch, 1979). These studies were conducted using surface electrodes.

Blood must circulate through the muscle during exercise to prevent build-up of metabolities (Basmajian & DeLuca, 1985). However, once the voluntary isometric contraction force of a muscle exceeds 15% of its MVC, the circulation is severely restricted (Bigland-Ritchie & Woods, 1984). The blood flow is practically occluded when the voluntary isometric contraction force reaches 50% of a muscle's MVC (Bonde-Peterson, Monk & Nielson, 1975). The relative force that occludes a muscle also depends on the size of the muscle and the geometry of the fibers (Bigland-Ritchie, 1981; Luttgens & Wells, 1983). The diminished circulation prevents removal of the muscle metabolities and the acid environment remains.

There are several characteristics of motor unit organization that should be recognized when contrasting voluntary and electrically stimulated muscle contractions. During a voluntary contraction, the motor units are activated asynchronously. Asynchronous activity causes various motor units to be active and inactive at different times and discharge at different rates during the contraction (Basmajian & DeLuca, 1985). Since the motor units and even the fibers innervated by a single motor neuron are dispersed throughout the muscle, this asynchronous behavior yields a smooth contraction (Bigland-Ritchie, 1983). Because individual motor units are not continuously firing, the rate variation may decrease fatigue. On the other hand, electrical stimulation activates a restricted number of motor units synchronously because of the location of the electrodes, current amplitude, and the stimulating frequency. This may lead to an increased rate of fatigue.

In a voluntary contraction, the small fatigue resistant Type I fibers are activated first. As more force is needed, the rate of firing

of these units is first increased and if more force is needed, the larger, more powerful Type II units are recruited. This sequence of activity is commonly known as the "size principle of Henneman" (Henneman, 1965). During electrical stimulation, there is a reverse of the "size principle" and the Type II fibers are activated first (Appell, 1988). The Type II fibers are innervated by large diameter axons that have a relatively low threshold to electrical stimulation (Solomonow, Eldred, Tyman & Foster, 1981). Benton, Baker and Bowman (1931) ties the greater rate of fatigue noted with electrical stimulation versus voluntary activity to the synchronous and preferential stimulation of the large diameter axons. This increased rate of fatigue was also noted by Currier and Mann (1983).

Many of the studies cited have implied that fatigue during a voluntary muscle contraction is caused by failure at a particular site in the chain of events that starts with excitation of the higher motor centers and culminates with the interaction of actin and myosin. However, voluntary muscle activity depends upon the proper functioning of the entire chain and disruption of the chain may occur at one site or at several sites simultaneously (Bigland-Ritchie, 1981). Because of variations in the motor unit activation pattern and the range of frequencies used to induce an electrically stimulated contraction, the site of muscle fatigue may be different when voluntary and electrically induced muscle fatigue are compared.

Functional Electrical Stimulation

In addition to the utilization of electrical stimulation to prevent muscle atrophy and strength loss, there has been considerable research into the use of this modality to assist patients in improving functional

activities following injuries to the peripheral and central nervous systems (Baker, Yek, & Wilson, 1979; Liberson, Holmquist, Scott & Dow, 1961; Packman-Braun, 1988; Solomonow, Eldred, Tyman & Foster, 1981; and Waters, McNeal & Perry, 1979). Solomonow, et al. (1982) and Packman-Braun (1988) cite the high rate of muscular fatigue and an inability to achieve finely graded contractile tension as the main drawback to the use of electrical stimulation as a functional tool.

The Electromyographic Signal as an Indicator of Muscle Fatigue

Changes in the electromyographic signal have been used as an indicator of muscle fatigue for over twenty years (Broman, Bilotto & DeLuca, 1985; Gatev, Ivanova & Gantchev, 1986; Horita & Ishiko, 1987; Kadefors, Kaiser & Petersen, 1968; Komi & Tesch, 1979; Kranz, Williams, Cassell, Caddy & Silberstein, 1983; Lindstrom, Magnusson & Peterson, 1970; Mills, 1982; Shochina, Gonen, Vatine, Mahler & Magora, 1986; Zwarts, Van Weerden & Haenen, 1987). The current literature describes a slowing of recorded EMG frequencies as a muscle fatigues (Broman, et al., 1985; Horita, et al., 1987; Kranz, et al., 1983; Zwarts, et al., 1987). An alternative method for analyzing local muscle fatigue was described by Hagg (1981). This author described a method of counting the number of zero crossings of the EMG signal and postulated that a decrease in the number of zero crossings was indicative of muscle fatigue. Inbar, et al. (1981), Masuda, Meyano and Sadoyama (1982), and Basmajian and DeLuca (1985) also describe this method of measuring changes in the frequency spectrum by counting zero crossings of the EMG signal. Lindstrom, et al. (1977) theorized that the slowing of recorded EMG frequencies seen in muscles fatigued by voluntary activity was due to a decrease in the conduction velocity of the muscle fiber (sarcolemma). Recent studies

agree with Lindstrom's hypothesis (Broman, et al., 1985; Horita & Ishiko, 1987; Komi & Tesch, 1979; Kranz, et al., 1983; Zwarts, et al., 1987). These authors speculate that slowdown in the conduction velocity of the sarcolemma is caused by the accumulation of metabolities -- especially lactic acid -- in the muscle. These authors conclude that the decreased intracellular pH is mainly responsible for the decrease in the muscle fiber conduction velocity. Hagg (1981) theorizes that the decrease in conduction velocity is directly related to the decrease in the number of zero crossings seen in the EMG signal during muscle fatigue. Nitz (1984) cites the work of Fowler, et al. (1972) who speculate that changes in the area under the negative portion of the compound muscle action potential recorded during electromyography are the most accurate instruments to quantify the number of active muscle fibers during a contraction. A decrease in the number of active fibers results in a decreased area and may be indicative of muscle fatigue.

As the force of a muscle contraction increases, there is a change in the type, number and rate of motor units firing. As increased resistance is encountered by a muscle, the force of contraction is increased by either increasing the number of active motor units or by increasing the rate of firing of the already active units. Since the EMG signal is a reflection of the electrical activity of the contracting muscle and the electrical activity is determined by the number of motor units recruited and their firing frequency, there is a direct relationship between the EMG signal and the force generated by the contracting muscle (Basmajian & DeLuca, 1985, Bigland-Ritchie, 1981). Basmajian and DeLuca reviewed the literature from 1952 to 1979 and found researchers reported a linear or

quasilinear relationship between force and EMG (Basmajian & DeLuca, 1985). The non-linear component of the force/EMG relationship occurs at force levels below 20% MVC. The non-linearity may be because of the relative location of the slow-twitch and fast-twitch muscle fibers within the muscle (Basmajian & DeLuca, 1985). Hultman and Sjoholm (1983) reported that during a fatiguing electrical stimulation to the human quadriceps femoris muscle, the amplitude of the recorded EMG signal and the muscle force decrease at the same rate. Although there is considerable variation between muscles, Clamann (1970) suggests that the majority of fast-twitch muscle fibers are located in the muscle's surface. Edgarton, et al. (1975) and Johnson, Polgar, Weightman and Appleton (1973) also reported uneven distribution of fiber types in human muscles. Saltin and Gollnick (1983) report a tendency for fast-twitch muscle fibers to be more superficial, while Armstrong (1984) -- examining the rat hindlimb -- also found the fast-twitch fibers to dominate in the superficial areas. Other authors speculate that two joint muscles such as the rectus femoris may have an increased percentage of fast-twitch muscle fibers when compared with muscles crossing only one joint (Garrett, Seaber, Glisson and Ribbeck, 1987). Bigland-Ritchie (1981) suggests than uneven fiber distribution may account for the non-linear surface EMG/force relationship at low force levels. The smaller Type I units, recruited early in a graded contraction, are located deep in the muscle and would yield smaller EMG signals because of distance from the recording electrode. The force level at which new motor units are recruited varies from muscle to muscle. In the Type I dominant first dorsal interossi and adductor pollicis muscles, all motor units are recruited by 30 to 50% MVC (Milner-Brown, Stein & Yemm, 1973; Stephens &

Usherwood, 1975). Garnett, O'Donovan, Stephens and Taylor (1978) reported that only fast-twitch fibers were recruited past 30% of the MVC in the mixed fiber type gastrocnemius muscle. In the biceps brachii -- a muscle of mixed Type I and II fibers -- new motor units are recruited up to 85% MVC (Kukulka & Clamann, 1981). Petrofsky (1982) reported that by the time a muscle reaches 50% of its MVC, all the motor units are recruited. Any increase in muscle tension beyond this point is due to increased motor unit firing.

The fast-twitch fibers are larger than the slow-twitch fibers and produce a higher frequency firing rate and a larger EMG signal amplitude (Bigland-Ritchie, 1981; Basmahian & DeLuca, 1985). Muscles with a high proportion of fast-twitch fibers also produce more muscle torque and fatigue more easily than muscles composed mostly of slow-twitch fibers (Komi & Tesch, 1979). Thorstensson, Larsson, Tesch and Karlsson (1977) reported that athletes with a high proportion of Type II muscle fibers were able to generate more torque than those with Type I dominant muscles (quadriceps femoris tested). Muscles with high percentages of fast-twitch fibers show a more pronounced shift toward the low frequency spectrum (recorded with surface EMG) during muscle fatigue (Viitasio & Komi, 1978). Once the fast-twitch fibers are recruited, the relationship between force and the surface EMG signal becomes linear (Bigland-Ritchie, 1981). Woods and Bigland-Ritchie (1983) report that a linear relationship between force and EMG occurs at 30% MVC in muscles of mixed fiber type. The decrease in intracelluar pH seen during exercise can cause a decrease in the muscle membrane conductivity with a subsequent decrease in the conduction velocity (Jennische, 1982; Orchardson, 1978). This decrease in conduction velocity has been reported to cause the

decreased frequency observed using surface EMG (Kranz, et al., 1983; Broman, et al., 1985; Zwarts, et al., 1987; Horita, et al., 1987), while Mills (1982) speculates that as fatigue progresses the fast-twitch fibers cease to fire, causing the spectral shift. Mills suggests that the fast- and slow-twitch fibers make up different areas of the surface EMG spectrum and as the fast-twitch fibers fatigue and drop out, the spectrum slows. Hultman and Sjoholm (1983) speculate that during electrical stimulation of skeletal muscle, the Type II fibers cease firing early in the contraction (because of their low resistance to fatigue) causing a decrease in the amplitude of the EMG signal. Komi and Tesch (1979) speculate that muscles with predominate fast-twitch fiber composition fatigue more rapidly than slow-twitch dominant muscle because they produce more lactic acid during comparable muscle activity. These authors report a positive relationship between decreased torque, percentage of fast-twitch fibers in the muscle, and a shift to lower frequencies recorded by the surface EMG during fatigue. Mills (1982) ties the shift directly to metabolic changes in the muscle. Cabric, et al. (1988) postulates that electrical stimulation of a muscle has a more pronounced effect on fast-twitch fibers than on slow-twitch components.

Pain Associated with Electrical Stimulation

Halbach and Straus (1980) maintain that the pain and discomfort associated with the clinical use of electrical stimulation are the major limitations to the use of this modality for strength training. Type II muscle fibers are associated with strength while Type I muscle fibers are dominant in endurance activities (Astrand & Rodahl, 1977). Cabric, et al. (1988) reported that when electrical stimulation is used to increase muscle strength, the current must be of sufficient strength to produce a

strong fused tetanic muscle contraction. These authors also showed that electrical stimulation at relatively high intensities affect the Type II fibers more than the Type I fibers. Parker (1986) tied the rapid force rise seen during electrical stimulation to predominent Type II fiber activation. Knuttgen (1976) stated that the most effective means of increasing muscle strength is to exercise at 100% of the MVC and a minimum of 50% MVC is necessary to achieve a training effect. DeVries (1980) reports that 35% of the MVC is sufficient to reach a training threshold. Selkowitz (1985) cites the work of Kots, who reported using electrical stimulation at 30% greater than an individual's MVC to achieve strength gains. Kots reported no pain with stimulation of this magnitude (Kramer, 1987). Owens and Malone (1983) reported subject discomfort while training their subjects at 60% of their MVC and Currier and Mann (1983) reported unpleasantness. These authors also noted than when voluntary muscle activity was superimposed upon the electrically stimulated activity, the discomfort decreases. These studies used a 2,500 Hz carrier frequency modulated at 50 bursts. Using a similar current, Laughman, et al. (1983) reported no pain in patients being treated for patellofemoral problems. However, the patients were only being stimulated to 33% of their MVC. Cabric, et al. (1988), stimulating to patients tolerance, reported no skin discomfort when using 2,500 Hz carrier frequency. When stimulating subjects to their maximal tolerance, Kramer (1987) reported increased skin sensitivity at lower frequencies (20 Hz). McMiken (1983) reported little or no pain in subjects stimulated at 30% MVC (at 70 Hz). Vodovnik, et al. (1965) and Crochetiere, et al. (1967) found that they could affect subjects' pain perception by varying the frequency of the electrical stimulator.

Vodovnik, et al. (1965) reported a decrease in pain perception with frequencies greater than 500 Hz when a sinusoidal wave form was utilized, while Crochetiere -- using a square wave -- found frequencies about 300 Hz to be most comfortable. Kots suggested that 2,200 to 2,500 Hz modulated current has an anaesthetic effect on the skin and this allows a more intense motor stimulation (Kramer, 1987). Maximal muscle force has been shown to occur at stimulation frequencies ranging from 70 to 90 Hz (Edwards, Young, Hosking & Jones, 1977; Moreno-Aranda & Seireg, 1981).

Strength duration curves have been used to demonstrate selective stimulation of sensory, motor, and pain fibers. By adjusting the pulse duration of the stimulating current, specific neurological responses can be targeted (Alon, 1987). The peripheral nerves are classified according to their conduction velocity (Li & Bak, 1976). The thinly myelinated A-delta fibers and the unmyelinated "C" afferents are thought to conduct painful stimuli (Perl, 1968; Collins, Nulsen & Randt, 1960). Higher frequency electrical stimulation is also useful in decreasing impedance to current flow (eg, 2500-4000 Hz). The higher the frequency, the lower the tissue impedance. These higher frequencies facilitate current flow across the electrode-skin interface (Cook, 1987). The greater the skin resistance, the more superficial (less penetrating) the stimulation. Since motor fibers are generally located deeper than pain sensitive fibers (DeGirardi, Seaborne & Goulet, 1984; Melzak, 1975), higher frequency stimulation may decrease the activity of pain fibers (Alon, 1987).

Muscle Soreness Associated with Exercise

Individuals engaged in unaccustomed exercise or when resuming training after a layoff from training, often experience muscle pain. The

pain is of two types and results from different etiologies. Acute muscle soreness experienced during or immediately after vigorous exercise is thought to be due to muscular ischemia (Francis, 1983). With ischemia, the usually diffusable end products of exercise (lactic acid and potassium in particular) accumulate and are thought to stimulate pain receptors in the muscle. Francis further states that this soreness is not long lasting and begins to subside as soon as the ischemic condition is resolved. A second type of muscle soreness frequently occurs 24 to 48 hours after an exercise bout. This type of soreness is often referred to as "delayed muscle soreness." Garrett (1985) credits Hough (1902) with first describing and investigating this phenomenon. He reported that this condition was associated with high intensity exercise and that sudden contractions or jerking motions were associated with the delayed onset of muscle soreness. Later studies by Asmussen (1956) and Abraham (1977) are in agreement with the work of Hough (Francis, 1983: Garrett, 1985). Abraham (1977) reports increased levels of hydroxyproline in the urine of individuals suffering delayed muscle soreness. Hydroxyproline is a component of collagen and high levels of this amino acid in the urine have been reported to indicate the rate of collagen breakdown (Garrett, 1985). Schwane, Watrous, Johnson and Armstrong (1983) reported that high levels of blood lactic acid -- thought to be a cause of acute muscle soreness during and following exercise -- are not related to delayed muscle soreness. With delayed muscle soreness, the majority of microscopic changes noted in the muscle fibers are in the Type II fibers (Friden, Sjostrom & Ekblom, 1981). The greatest amount of delayed muscle soreness is associated with eccentric exercise (Francis, 1983). McCully and Faulkner (1985) have demonstrated that intense eccentric exercise

causes severe muscle damage while isometric and isokinetic exercise do not. Cote, et al. (1988) confirm this observation and state that damage done to the muscle fibers during the eccentric phase of muscle contractions is responsible for the observed muscle soreness.

Summary

Muscle fatigue is a complex process; the cause of which has been debated for many years. Researchers have attempted to isolate a specific cause of muscle fatigue without consensus. Recent studies indicate that muscle fatigue induced by voluntary activity may be caused by events that occur simultaneously or in succession from the initiation of muscle activity in the brain to the interaction of actin and myosin at the cellular level. During a voluntary muscle contraction, there is asynchronous motor unit activity which reduces the rate of muscle fatigue. However, during the electrical stimulation of muscles a synchronous contraction is observed. This synchronous motor unit activity hastens the rate of muscle fatigue and may limit the use of this modality as a training device. In addition to the increased rate of muscle fatigue reported during electrically induced muscle contractions, the pain associated with electrical stimulation has limited the clinical use of this modality. Several researchers have reported that by manipulating the frequency of the stimulator, a subject's pain perception can be altered.

Muscle fatigue has been tied to decreases in torque production as well as changes in the activity of surface EMG. During prolonged maximal muscle contractions, there are changes in both torque production and in the recorded EMG activity. These changes have been tied to the type and number of motor units activated as fatigue occurs. Researchers have

quantified these changes by using torque curves, counting zero crossings of the EMG signals, and by measuring the area under the negative deflection of the EMG curve.

CHAPTER 3

METHODS AND PROCEDURES

This chapter describes the procedures involved in the present study. The sections included are: (a) subjects, (b) procedures, and (c) statistical analysis.

SUBJECTS

Selection of Subjects

The recruitment of subjects for this study began in February, 1988. The recruitment consisted of visiting classes and explaining the experimental procedure and asking interested individuals to volunteer. Pamphlets requesting volunteers were also placed in student mailboxes. The majority of the subjects were drawn from the junior and senior classes of the Department of Physical Therapy and the Department of Health, Physical Education and Recreation at the University of Kentucky. All subjects were informed of the testing procedures, purpose of the study, and risk factors of the study before giving written consent. The study and consent form were approved by the Biomedical Human Investigations Review Committee (Appendix A). Twenty-eight volunteers participated in the study. One subject was dropped from the study when she was unable to tolerate electrical stimulation at the required amplitude. Subjects ranged in age from 18 to 35 years, and had a mean height of 176.6 cm., and mean weight of 71.5 kg. Table 1 displays demographic data. Each subject received twenty-five dollars after completion of the study. Funds were obtained from a grant from the Kentucky Chapter of the American Physical Therapy Association.

Table 1

Demographic Data of Subjects Who Participated in This Research Project

	Weight (kg)	Height (cm)	Age (yrs)
$\bar{X}$	71.5	176	22
SD	9.6	7.5	2.2

PROCEDURE

This study was carried out in two phases. Phase I determined the combination(s) of bursts and carrier frequencies during electrical stimulation that satisfied the following criteria:

1. Produced an equivalent of 50% of each subject's maximum voluntary contraction (MVC), and
2. Produced the least amount of discomfort in subjects being stimulated at 50% of their MVC.

Fulfillment of these combined conditions is considered the optimum for clinical electrical stimulation. Phase II, using information about optimum frequency obtained in Phase I, determined the effect of incrementing the current amplitude on muscle fatigue and torque production. The current amplitude of the electrical stimulation was incremented after every series of two muscle contractions. The periodic incrementation of current amplitude is an accepted clinical practice in physical therapy but its effect on muscle contraction and performance has not been documented in the literature.

Phase I

Familiarization Session. Subjects participated in a familiarization session to reduce the effect of learning on test scores. At that time, subjects' height, weight, sex, and age were recorded. Edwards et al. (1977) reported that the force of a MVC of the quadriceps femoris muscle was proportional to a subject's body

weight (r = .92). These authors predicted a "normal" MVC to be 75% of body weight and that the "lower limit of normal" (the third percentile) was 50% body weight. The results of female subjects fell close to the male values. Hultman et al. (1983) and Edwards (1984) also reported a linear relationship between body weight and the MVC of the knee extensors. Any subject whose MVC was not within 75% of their body weight was eliminated from this study. This screening was to ensure that subjects were sufficiently motivated to participate.

The subject's maximum isometric knee extensor torque (the highest torque reading of three repetitions was used as the maximum) was determined using the Cybex isokinetic dynamometer, 50% of this maximum was used as a target torque during electrical stimulation. These values were determined during the familiarization session and used throughout the remainder of this study. The isometric knee extensor torque was recorded using a Cybex II isokinetic dynamometer (Cybex Division of Lumex, Inc., Ronkonkoma, New York 11779) set at 0° angular velocity (Figure 1). The dynamometer was calibrated per manufacturer's instructions before the experiment began. Kramer (1987) reported that the reliability of the Cybex II varied between .96 and .98, while Clarkson et al. (1982) reported test-retest reliability of the Cybex II to be r = .97. A damp setting of 3 was used throughout this study. Damping modifies overshoot and oscillation of the recording stylus by slowing the stylus response. Damping thus provides a smoother recording of the torque production (Bemben, Grump and Massey, 1988). Subjects were seated in the test chair with their hips extended to 120° and the knee of their left lower extremity flexed to 60° (Figure 2). This position was used to

Figure 1 - Cybex II isokinetic dynamometer and dual channel recorder.


CYBEX II

Figure 2 - Subject seated with the left knee secured at 60° flexion. The dynamometer is in the isometric mode (0°/sec) and the surface electrode is in place.


NEXT

maximize the isometric knee extensor torque (Currier, 1977, 1979, 1982; Clarke and Bailey, 1950; Clarke, Elkins and Martin, 1950). Body stabilization was provided by a lap seat belt and a back support. The axis of rotation of the Cybex II dynamometer was adjusted to the anatomical axis of the knee joint and the angle of flexion of the knee and hip was measured manually using a manual goniometer. The lever arm of the Cybex II was kept consistent for each subject by placing the inferior border of the ankle pad immediately superior to the medial malleolus (Epler, Nawoczenski, Englehardt, 1988) (Figure 3). The Cybex II dual channel recorder was used to record the torque generated by the knee extensors (Figure 1).

Following the torque measurement(s) taken during the familiarization session, each subject was given sufficient time to familiarize himself with the Electrostim 180-2i high intensity electrical stimulation unit (Figure 4). The Electrostim 180-2i uses surface electrodes. Two 12.5 x 8 centimeter electrodes served as the stimulating electrodes during the study (Figure 5). One electrode was placed over the vastus medialis muscle 4 centimeters proximal to the superior border of the patella and slightly medial to the medial border of the patella. The other electrode was placed on the skin over the vastus lateralis muscle on the middle one-third of its muscle belly (Figure 6). The carbon-silicone electrodes were separated from the skin by water soaked sponges which served as coupling media. The flexibility of these electrodes assured total contact with the muscle as it changed shape during the electrically stimulated contraction. The electrodes were held in place by elastic straps. Prior to stimulation, the electrode impedance was recorded to test the quality

Figure 3 - The ankle pad of the Cybex II was placed immediately superior to the medial malleolus to assure a consistent lever arm length.

Figure 4 - The Electrostim 180-2i High Intensity Electrical Stimulation Unit.

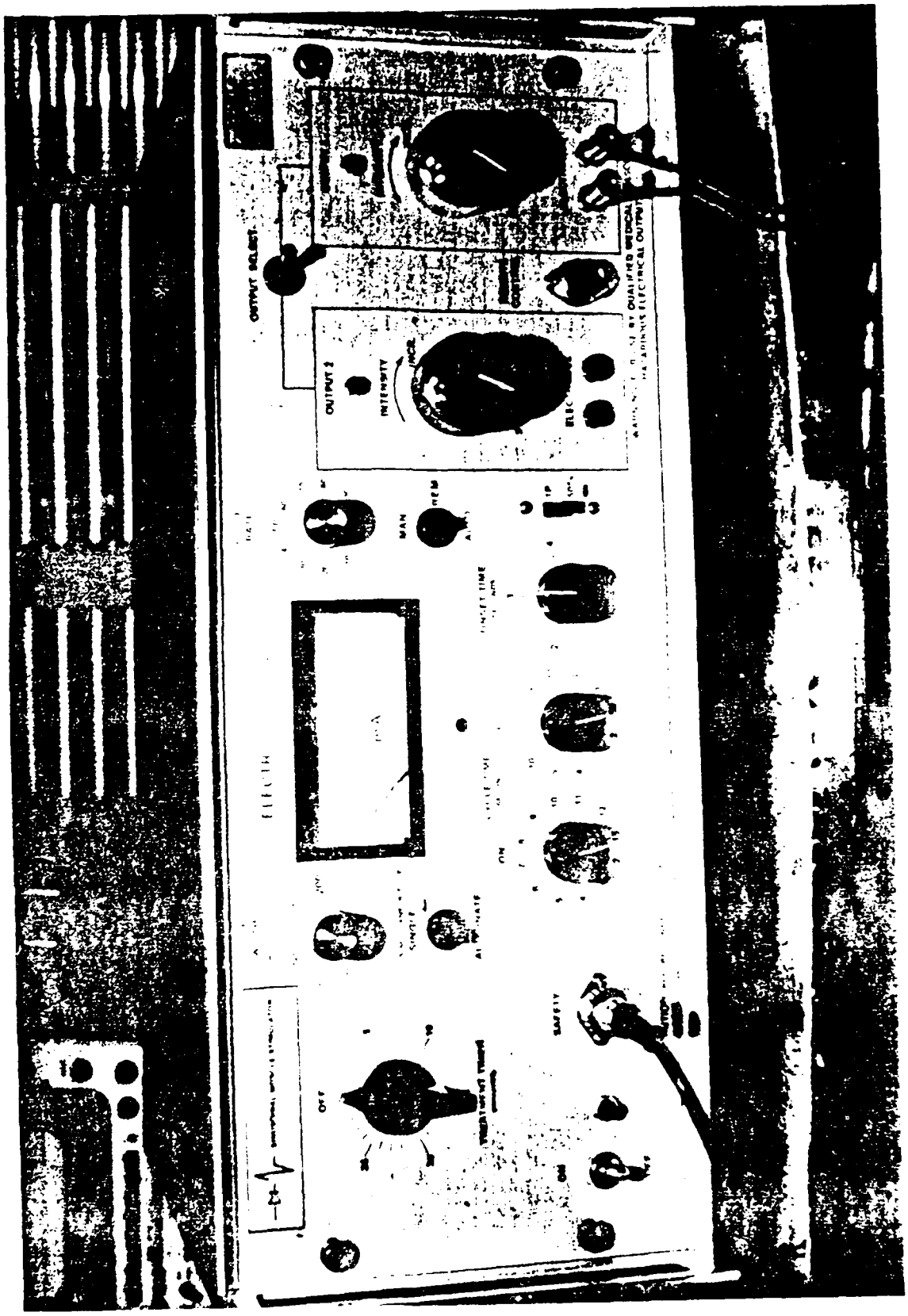

OUTPUT SELECT
OUTPUT 2
INTENSITY
MAIN
SAFETY

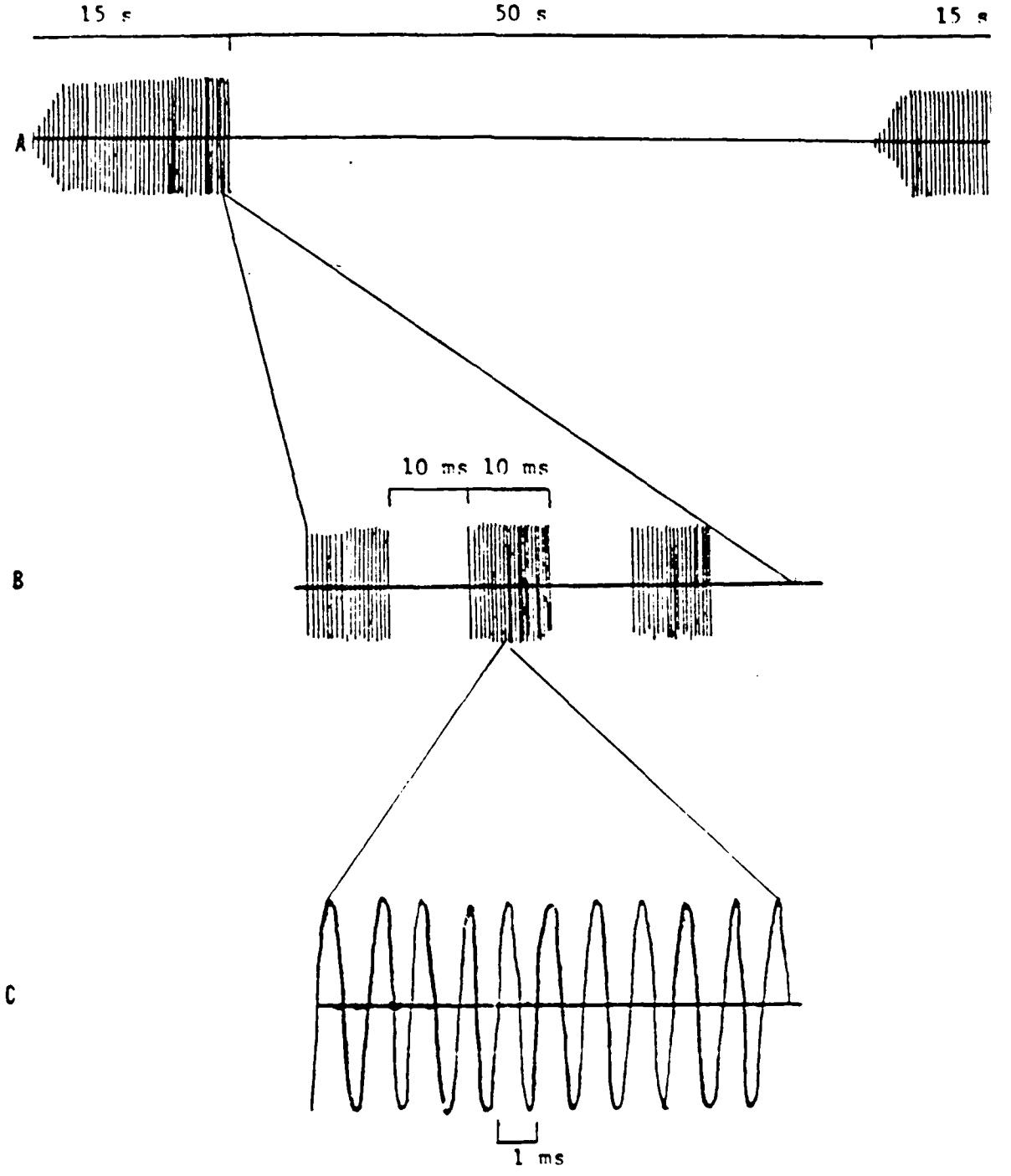
Figure 5 - Subject's left thigh with two 12.5 x 8 centimeter electrodes in place.

of the electrode attachment and skin preparation. An autogen 1700 biofeedback device was used to test impedance and an impedance level of less than 50,000 ohms was considered arbitrarily acceptable to proceed with the electrical stimulation testing (Autogen 1700 testing manual). The Electrostim 180-2i high intensity electrical stimulation unit delivered 15 seconds of electrical stimulation in the form of bursts that were repeated at 50Hz, 70Hz, and 90Hz. The 15 seconds electrical stimulation had a ramp on time of 3 seconds and maintained a peak amplitude (pulse charge) for 12 seconds. The current amplitude (pulse charge = pulse amplitude x duration) or current was abruptly stopped following the 15 seconds of stimulation (0 ramp off) and a 50-second rest period (interburst interval) followed. This 1:3 on/off ratio was maintained throughout the experimental protocol. The shape of each pulse within the bursts was a sine wave which repeated at a rate of 2,500Hz, 5,000Hz or 10,000Hz (carrier frequencies) (Figure 6). Subjects were encouraged during the familiarization session to volitionally contract their quadriceps femoris muscles with an intensity sufficient to produce the equivalent of 50% of their maximal knee extensor torque and were told that this would be the level of stimulation they would receive during the experimental session. The purpose of this practice session was to minimize any learning associated with the testing procedure and to allow the subjects to experience a variety of stimulating frequencies. Subjects were instructed to refrain from voluntarily contracting the quadriceps femoris muscle when being electrically stimulated (torques were monitored by the Cybex dynamometer). Subjects were not tested for torque until they indicated that they felt accustomed to the electrically induced contraction. Individual subjects produced greater or less than

Figure 6 - (a) An example of the 50 Hz burst mode and 2,500 Hz carrier frequency during a 15 second stimulation followed by 50 seconds of rest a three second ramp on time is utilized.

(b) The 50 Hz burst mode

(c) Sine wave


15 s
50 s
15 s
A
10 ms 10 ms
B
C
1 ms

50% of their maximum torque during the familiarization session depending on individual adjustment to electrical stimulation. However, 50% of the maximum torque was a target and minimum acceptable score during the experimental phase of this study. Since changes in the characteristics of the electromyographic (EMG) signal were used to quantify the fatigue of the quadriceps femoris muscle following stimulation, an EMG was taken during the familiarization session to introduce each subject to this procedure.

Experimental sessions.

Following the familiarization session, there were three experimental sessions during Phase I.

During each of these three experimental sessions, each subject randomly received one of three predetermined burst frequencies (50; 70; or 90Hz) combined with a randomly chosen carrier frequency (2,500, 5,000, or 10,000Hz). Combinations were determined using random number tables. Each subject received a series of ten electrical stimulations at an amplitude sufficient to produce approximately 50% of their maximum voluntary torque as measured on the first contraction. This uniform amplitude of electrical stimulation was chosen so that the amount of muscle fatigue (torque decrement/electrically induced contraction) with each current combination could be determined. The number of milliamperes necessary to produce 50% of each subject's MVC at each frequency combination was recorded. Following a five-minute rest period, each subject received another electrical stimulation bout using a randomized combination of carrier and burst frequency and the frequency-torque-fatigue relationship was again determined by assessing the recorded torque curves. During the five-minute rest

period, subjects were encouraged to walk around the Physical Therapy Research Lab or to actively flex and extend their knee several times while seated. McArdle, Katch and Katch (1986) reported that mild exercise during rest periods results in faster removal of lactic acid from exercised muscles (active recovery). Bigland-Ritchie's, et al. (1986) data show that a five-minute rest period is adequate to recover from fatigue during electrical stimulation. Basmajian and Deluca (1985) and Harris (1981) also report that 4 to 5 minutes of rest are adequate amounts of time to recover from a bout of localized muscle fatigue. Much longer periods of time are needed by subjects undergoing exercise to recover from systemic fatigue since large amounts of lactate would be present in the overall circulatory system (Sahlin, 1978). During the period of time of the localized muscle fatigue induced in this experiment, the increased lactate was expected to be primarily involved in the quadriceps femoris muscle and consequently a five-minute rest between bouts was considered to be sufficient for complete recovery from fatigue. During the course of three treatment sessions, each subject experienced all nine combinations of the predetermined bursts and carrier frequencies.

Immediately following the tenth contraction (stimulation induced) of each combination of electrical stimulation, the subject rated the pain perception using a visual analog scale (VAS). A ten-centimeter vertically oriented VAS was used. Each scale was drawn on a plain white 7.6 x 12.7 cm (3 x 5 inch) card. Subjects were asked to rate the pain or discomfort associated with the stimulation between the extremes of "no pain" and "pain as bad as it could be". The boundaries of the ten-centimeter scale were defined by a one

centimeter line (Scott and Huskisson, 1976; Huskisson, 1983). (Figure 7).

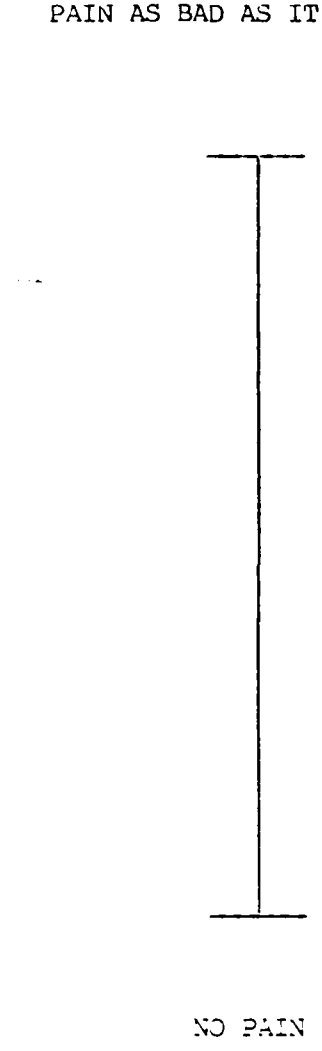
Adequate time (3-5 minutes) for warm-up was afforded each subject prior to each experimental session and consisted of each individual stretching his or her quadriceps femoris, hamstring and gastrocnemius-soleus muscle groups.

EMG Recordings.

Prior to the bout of 10 electrically stimulated muscle contractions, a 1 second surface electromyograph (EMG) of the quadriceps femoris muscle was taken in preference to using needle or fine wire electrodes which may cause intensive pain during high force muscle contractions (Gatev, et al. 1986). The pain associated with fine wire and needle electrodes also may effect the firing pattern of motor units (Datta and Stephens, 1981; Hennerz and Grimby, 1979).

Six EMG's were taken during each experimental session. The recording electrodes were placed over the vastus medialis muscle (5 centimeters) above the base of the patella and slightly medial to the medial border of the patella. The skin of each subject was marked to assure consistent surface electrode placement during the repeated EMG's necessary during each experimental session (Figure 9). Recording electrodes were placed on the same skin location, so that the same population of motor units were being recorded. These exact electrode placements allowed comparison of pre- and post-exercise EMG signals (Mills, 1982). The EMG signal is dependent upon the distance the surface electrodes are from the active muscle fibers. Any change in muscle volume or displacement of the muscle under the skin could cause variations in the EMG signal (Peres and Maton, 1987). Since

Figure 7 - Vertically oriented visual analog scale.



changes in muscle length can also affect surface EMG signals (Okada, 1987) joint angle positioning was constantly monitored for consistency using goniometry. The Cadwell 5200-A was used to record the EMG signals (Cadwell Laboratories, Inc., Kennewick, Washington 99336). This EMG unit allowed the recording of a visual representation of the surface EMG signal during both pre- and post-muscle stimulation. A 60 cycle notch filter was used to eliminate 60Hz power line noise. The high cut filter was set at 10,000 Hz and the low cut filter was set at 100 Hz as recommended by the manufacturer. A sweep speed of 20 milliseconds per division of the oscilloscope grid was used throughout the experimental protocol, as was an amplifier gain of 500uV. A bar electrode containing two 1 centimeter diameter recording metal discs placed 3 centimeters apart was used to record the EMG signal. Leads from the bar electrodes were plugged into the pre-amplifier of the EMG unit. Nitz (1984) and Fowler, et al. (1972) reported that the area under the negative portion of the compound muscle action potential (MAP) is an accurate method of assessing the relative activity of a muscle. The area under the negative portion of the MAP was measured using an image analysis system (Zeiss Video Plan Image Analysis System, Carl Zeiss, Inc., Thornwood, New York).

Phase II.

Determining the effect that incrementing the intensity of stimulation has on fatigue and torque production.

The optimum combination of frequency and carrier frequency of Phase I was applied to each subject. After every two contractions, the intensity of the stimulation was increased to maintain torque levels at the pretest 50% MVC levels and to determine the effect this

approach of incrementing stimulus intensity has on fatigue (Note: Recorded torque with each muscle contraction that is electrically induced decreases in amplitude or Newton-meters because of fatigue. The actual stimulus was therefore incremented after every two induced contractions to maintain 50% of MVC torque). Each subject received ten stimulations at the chosen frequency and the amplitude was increased after every two electrically induced contractions. There were 5 increments of amplitude during this procedure. Pre- and post-EMG signals were recorded as in Phase I.

Data Analysis.

Following Phase I, a Pearson's correlation coefficient was run to determine the relationship between body weight and maximal isometric torque production. Descriptive data were utilized to ensure that subjects MVC vs. body weight were within normal limits ("normal" MVC is considered to be at least 75% of body weight, Edwards, 1977). Torque/frequency graphs were analyzed to determine the optimal frequency combination that produced 50% of a subject's MVC and the VAS was used to find the combination rated most comfortable by each subject. A two-way ANOVA with repeated measures (bursts and carrier frequency) was used to analyze the data. The interaction of bursts and carrier frequency on pain, torque, EMG charges, and milliamperage necessary to produce 50% of a subject's MVC were analyzed using a SAS statistical package. If the "F" value of the ANOVA was significant and there was no interaction between variables, a Duncan post hoc analysis was used to further differentiate the data. The changes in torque and EMG following stimulation were compared using a t-test.

The Phase II data were analyzed using a frequency distribution to determine which combination of bursts and carrier frequency was preferred by each subject. Descriptive statistics were used to analyze the percentage increase in milliamperage necessary to maintain 50% of a subject's MVC. A dependent t-test was utilized to analyze the differences in Phase I and II EMG changes when stimulating at the preferred frequency combinations (Recall that Phase I used a constant milliamperage during the ten stimulations, while in Phase II the milliamperage was increased 5 times during the ten stimulations). A significance level of .05 was used throughout this study.

Chapter 4

RESULTS AND DISCUSSION

This chapter begins with a statistical analysis of the results of this study and concludes with a discussion of these results. In the discussion section, the author will tie the results of this study to previous works and attempt to explain any unusual findings.

The results are divided into the categories Phase I and Phase II.

Phase I is subdivided into five sections and covers the following areas: (1) body weight and maximal torque production; (2) changes in torque following various combinations of electrical current bursts and carrier frequencies; (3) changes in the EMG signal following various combinations of electrical current bursts and carrier frequencies; (4) the milliamperage necessary to produce 50% of a subject's MVC at various combinations of electrical current bursts and carrier frequencies; and (5) the subject's rating of pain at the various combinations of electrical current bursts and carrier frequencies.

Phase II is divided into three sections. Section (1) analyzes the frequency combinations chosen least and most painful by each subject. Section (2) presents the percentage increase in milliamperage necessary to maintain 50% MVC at the frequency combination chosen most comfortable during Phase I. Each subject received 10 electrically induced contractions of the quadriceps femoris muscle at their preferred frequency combination. The final section, (3), analyzes the differences in Phase I and II EMG changes when stimulating at each subject's preferred frequency combination.

Phase I - Body Weight and Maximal Torque Production

All of the subjects participating in this study were able to

generate a maximum isometric torque on the Cybex II dynamometer of at least 75% of their body weight (Appendix B). The maximal torque ranged from 75 to 112% body weight in which Pearson product moment of r = .736 was significant at p = .001 (Figure 8).

Changes in Torque During the Nine Combinations of Bursts and Carrier Frequency

A t-test revealed a significant decrease in torque production following electrical stimulation when all nine combinations of electrical current bursts and carrier frequencies were linked. The mean decrease in torque was 31.8 N.m (SEM = 1.05) and scores ranged from a maximal decrease of 36.2 N.m (SEM = 4.19) at 70 bursts combined with a 2,500 Hz carrier frequency to a 23.2 N.m (SEM = 2.56) decrease in torque at 50 bursts combined with a 10,000 Hz carrier frequency (Tables 2 and 3). Figure 9 gives a graphic representation of these findings. The decrease in torque production following electrical stimulation was statistically significant (t = 30.15, p < .0001) when all combinations were linked or when combinations were viewed individually (Tables 2 and 3). When the decrease in torque was normalized (pre-stimulation torque minus post-stimulation torque, divided by the pre-stimulation torque), the mean percentage decrease in torque for all combinations of burst and carrier frequency was 30.3% (SEM = .9) (Table 4). An ANOVA reveals that bursts (50, 70, 90 Hz) did not significantly influence the decrease in torque (F = 1.20, p = .3042), but the carrier frequency had a significant impact (F = 12.05, p = .0001), (Table 5). A Duncan post hoc analysis shows no significant difference in the bursts (Table 5), but a significantly greater loss of torque when a carrier frequency of 2,500 Hz

Figure 8 - Correlation between subjects body weight (kg) and force produced (N). Pearson's product moment of r - .736 was significant at $\underline{p}$ = .001.

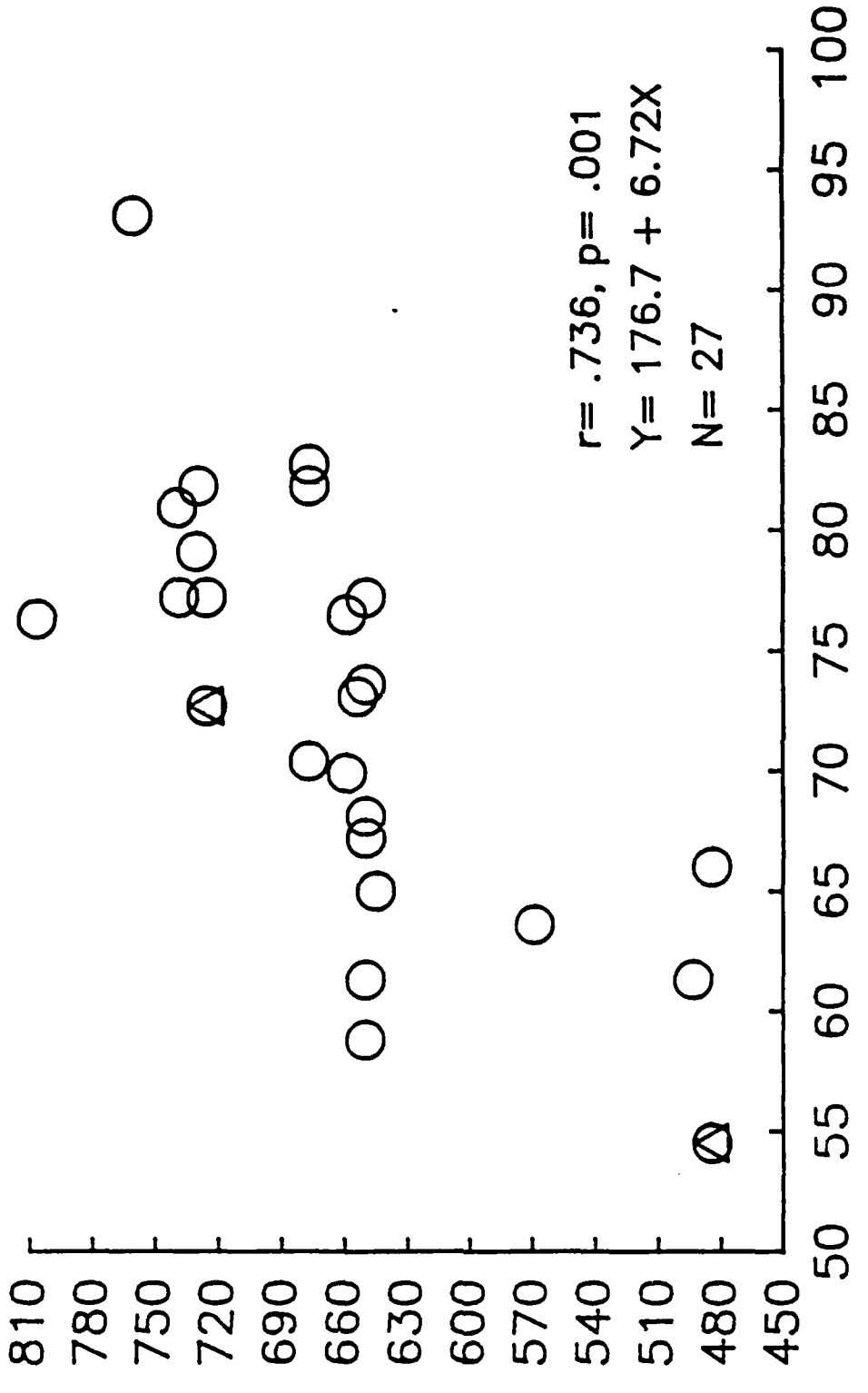

FORCE (N)
810
780
750
720
690
660
630
600
570
540
510
480
450
50
55
60
65
70
75
80
85
90
95
100
BODY WEIGHT (Kg)
r= .736, p= .001
Y= 176.7 + 6.72X
N= 27

Table 2

t-Test (Pre VS Post Stimulation) for Torque Changes (N.m) Following Nine Combinations of Bursts and Carrier Frequency

Source	N	$\overline{X}$	SEM	t	P
Decrease Torque	243	-31.8	1.05	30.15	0.0001

Table 3

t-Test (Pre VS Post Stimulation) for Torque Changes (N.m) at Individual Combinations of Bursts and Carrier Frequencies

Burst/Carrier Frequency	N	$\overline{X}$	SEM	t	P
50/2,500	27	34.5	3.2	10.82	0.0001
50/5,000	27	33.3	3.1	10.78	0.0001
50/10,000	27	23.2	2.6	9.03	0.0001
70/2,500	27	36.2	4.2	8.64	0.0001
70/5,000	27	34.6	2.9	12.03	0.0001
70/10,000	27	24.2	2.6	9.12	0.0001
90/2,500	27	34.6	3.1	11.12	0.0001
90/5,000	27	34.6	3.3	10.57	0.0001
90/10,000	27	31.1	2.5	12.19	0.0001

Figure 9 - Graphic representation of burst mode (50, 70, 90 Hz) and carrier frequency (2,500, 5,000, 10,000 Hz) to the torque decrement (N.m).

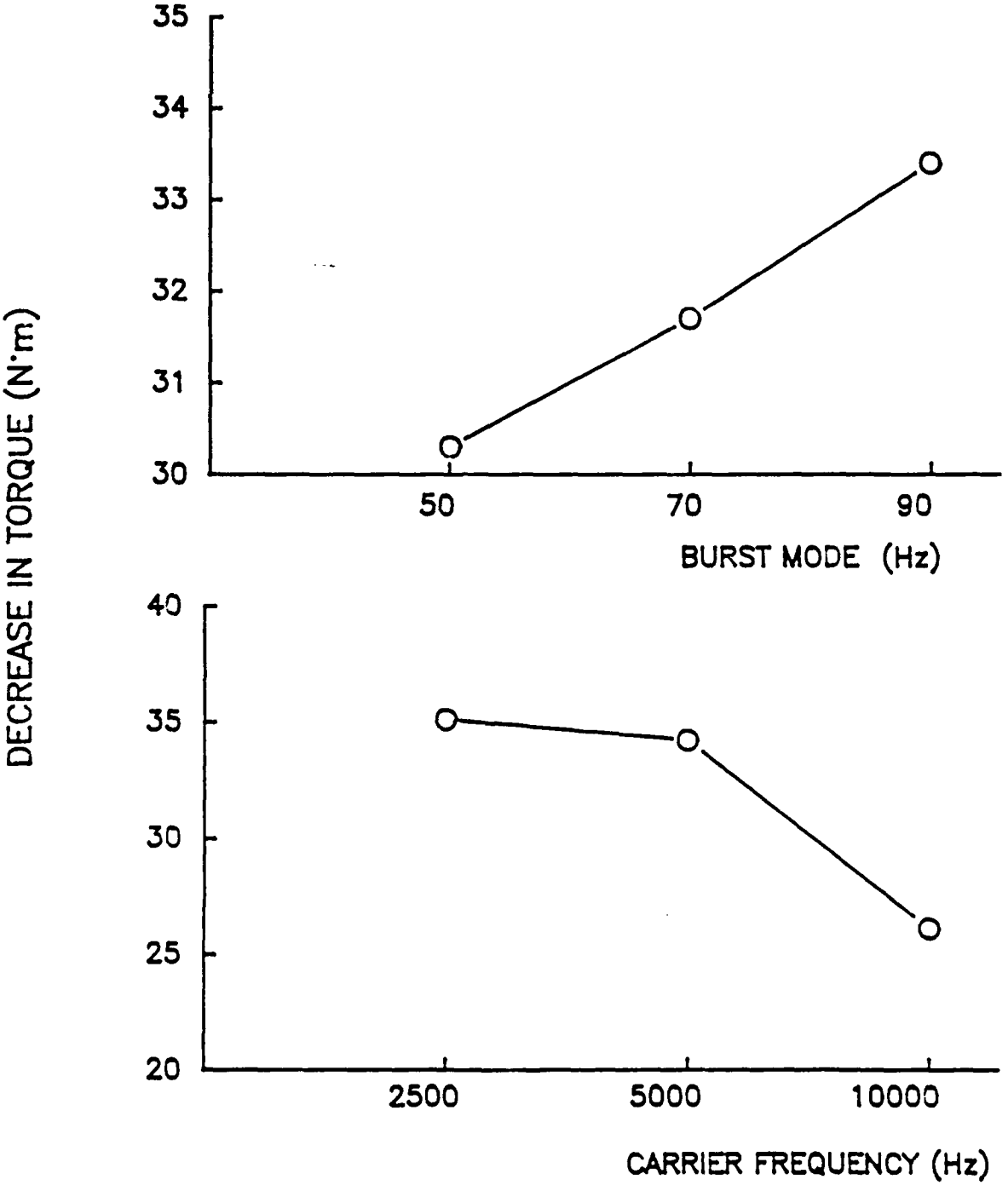

DECREASE IN TORQUE (N·m)
35
34
33
32
31
30
50
70
90
BURST MODE (Hz)
40
35
30
25
20
2500
5000
10000
CARRIER FREQUENCY (Hz)

Table 4

Normalized Torque Change (Pre VS Post Stimulation) Following Nine Combinations of Bursts and Carrier Frequencies

Source	N	$\overline{X}$	SEM
Normalized Torque	243	-30.3%	.9%

Table 5

ANOVA (bursts vs carrier frequencies) for Torque Change (N.m) Pre VS Post Stimulation

Source	df	SS	F	P
Model	34	31499.1	5.68	0.0001*
Error	208	33934.9	Mean Torque decrease = 31.8 N.m	
Burst	2	390.5	1.20	0.3042[A]
Carrier Frequency	2	3931.5	12.05	0.0001*
Burst*Carrier Frequency	4	680.7	1.04	0.3860[A]

[A] = non-significant

* A Duncan post hoc test revealed a significant difference between 10,000 Hz and 2,500 or 5,000 Hz carrier, but no significant difference between 2,500 Hz or 5,000 Hz.

($\overline{X}$ = 35.1) or 5,000 Hz ($\overline{X}$ = 34.2) were used as compared to 10,000 Hz ($\overline{X}$ = 26.1), (Table 5). There was no significant difference between the 2,500 and 5,000 Hz carrier frequencies. Figure 9 shows a trend of increasing torque loss as the burst mode is increased, and a reverse of this trend, as the carrier frequency is increased. Figure 10 depicts the combined effect of bursts and carrier frequency on torque. Figure 11 shows that when the burst mode is held constant, there is a decrease in the magnitude of torque decrement as the carrier frequency is increased.

Changes in the Area Under the Negative Deflection of the EMG Curve During the Nine Combinations of Bursts and Carrier Frequency.

A t-test revealed a significant decrease in the area under the negative deflection of the EMG curve when all nine combinations of burst and carrier frequency were linked. The decrease was statistically significant (t = 11.54, P = .0001). The mean decrease was 58.3 mm^2 (SEM = 5.1) and ranged from a maximum of 83.7 mm^2 at 70 bursts combined with a 2,500 Hz carrier frequency to 26.2 mm^2 at 50 bursts and a 10,000 Hz carrier frequency (Tables 6 and 7). A graphic representation of the combined affect of nine combinations of bursts and carrier frequencies on the change in area under the negative deflection of the EMG curve are shown in Figure 12. When the data were normalized (pre-stimulation EMG area minus post-stimulation EMG area divided by the pre-stimulation area), the overall mean percentage decrease in EMG area was 25.3% (Table 8). The ANOVA reveals a statistically significant interaction between burst and carrier frequencies (F = 2.61, P = .0364) and therefore the Duncan post hoc test could not be utilized to further differentiate the data

Figure 10 - Influence of the combined effect of burst mode (Hz) and carrier frequency (Hz) on torque (N.m).

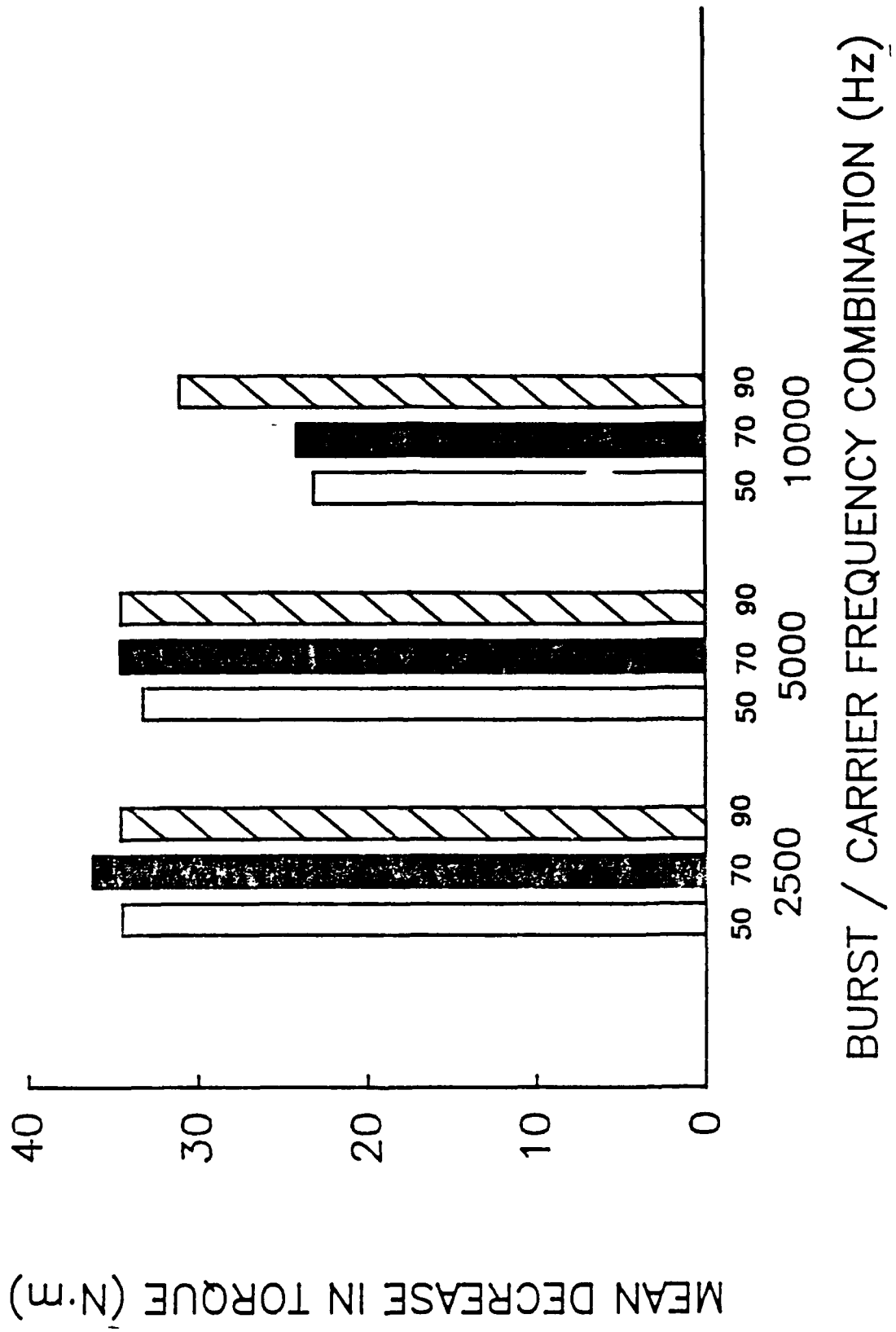

MEAN DECREASE IN TORQUE (N·m)
0
10
20
30
40
50 70 90
2500
50 70 90
5000
50 70 90
10000
BURST / CARRIER FREQUENCY COMBINATION (Hz)

Figure 11 - When burst mode is held constant, the decrease in the magnitude of torque decrement decreases as the carrier frequency (2,500, 5,000, 10,000 Hz) increases.

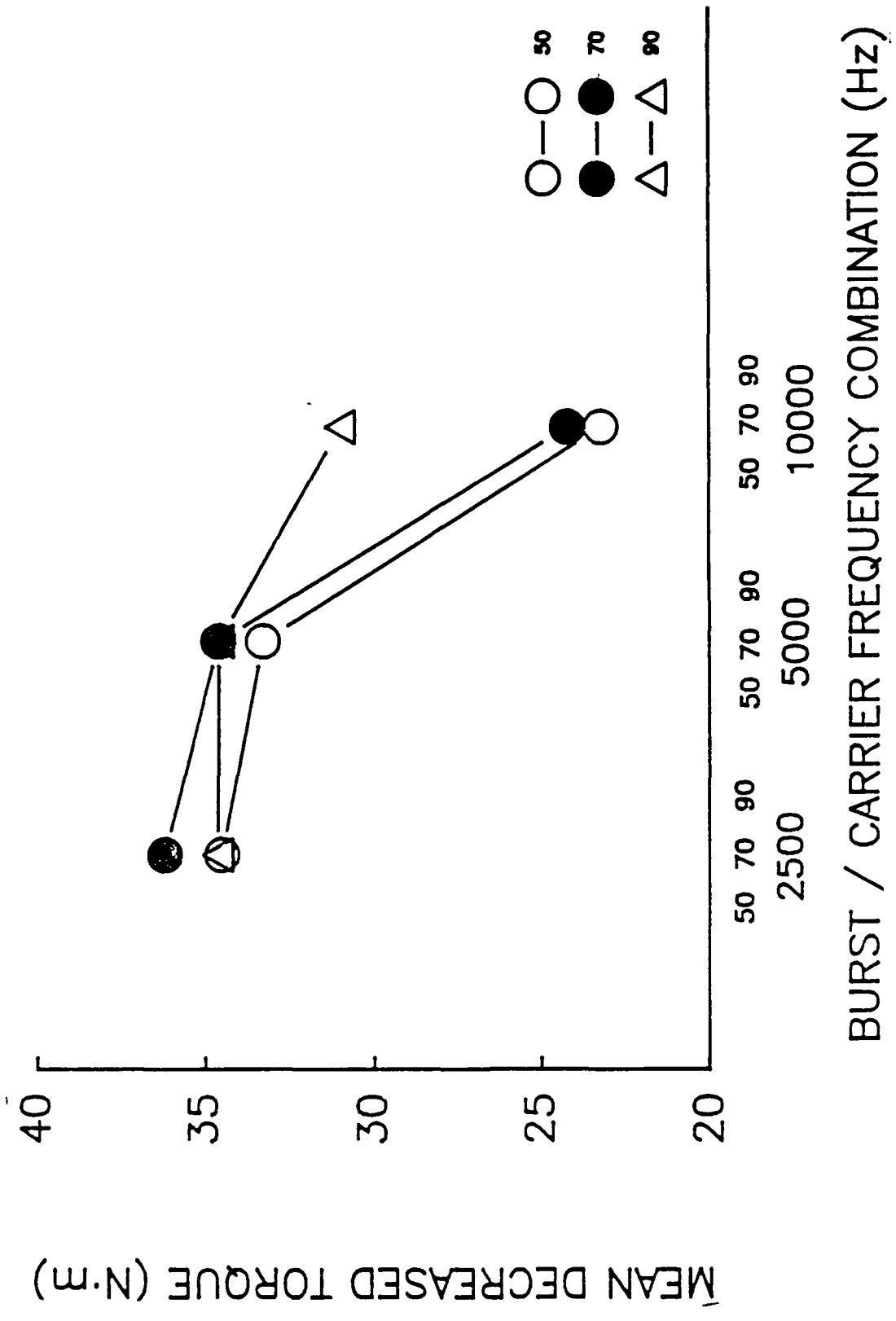

MEAN DECREASED TORQUE (N·m)
40
35
30
25
20
50 70 90
2500
50 70 90
5000
50 70 90
10000
BURST / CARRIER FREQUENCY COMBINATION (Hz)
50
70
90

Table 6

t-Test (Pre VS Post Stimulation) for EMG Changes (mm^2) Following Nine Combinations of Bursts and Carrier Frequencies

Source	N	$\overline{X}$	SEM	t	P
Decrease EMG	243	58.3	5.1	11.54	0.0001

Table 7

t-Test (Pre VS Post Stimulation) for EMG Change (mm^2) at Individual Combinations of Bursts and Carrier Frequencies

Burst/Carrier Frequency	N	$\overline{X}$	SEM	t	P
50/2,500	27	73.2	12.7	5.78	0.0001*
50/5,000	27	58.4	15.2	3.85	0.0007*
50/10,000	27	26.3	11.9	2.20	0.0371*
70/2,500	27	83.7	14.7	5.69	0.0001*
70/5,000	27	49.9	12.6	3.95	0.0005*
70/10,000	27	44.1	14.7	3.01	0.0057*
90/2,500	27	80.1	18.7	4.28	0.0002*
90/5,000	27	27.1	16.6	1.63	0.1147[A]
90/10,000	27	82.0	15.1	5.44	0.0001*

[A] = non-significant

* significant at $p = .05$

Figure 12 - Graphic representation of the affect of the nine combinations of burst mode (50, 70, 90 Hz) and carrier frequency (2,500, 5,000, 10,000 Hz) on the area under the negative deflection of the EMG curve.

MUSCLE ACTION POTENTIAL – AREA UNDER CURVE (mm²)
PRE vs POST STIMULATION DECREASE
0
10
20
30
40
50
60
70
80
90
50 70 90
2500
50 70 90
5000
50 70 90
10000
BURST / CARRIER FREQUENCY COMBINATION (Hz)

Table 8

Normalized (%) EMG Changes (Pre VS Post Stimulation) Following Nine Combinations of Bursts and Carrier Frequencies

Source	N	$\overline{X}$	SEM
Normalized EMG	243	-25.3%	3.8%

(Table 9). Figure 13 reveals a pattern of decreased loss of area under the negative deflection of the EMG curve as the carrier frequency increases in both the 50 Hz and 70 Hz burst modes. However, this pattern does not hold for the 90 Hz burst mode. There is a decrease in the loss of area under the compound action curves in the 90 Hz burst mode as the carrier frequency is increased from 2,500 Hz to 5,000 Hz, but the 90 burst/10,000 Hz carrier frequency breaks from this trend and shows a marked increase in EMG loss.

The Amount of Milliamperage Necessary to Produce 50% of a Subject's MVC at Various Combinations of Bursts and Carrier Frequency.

An ANOVA was used to analyze these data. Again, as with torque and EMG, the burst mode had no significant influence on the amount of mA needed for each subject to produce 50% of his MVC (F = 0.33, p = .7226) (Table 10). The carrier was statistically significant (F = 29.8, p = .0001), (Table 10). The Duncan post hoc analysis showed no significant difference in the mean milliamperage when the burst mode was tested (Table 10). However, the Duncan test revealed a significant difference in the mean milliamperage at all three carrier frequencies. A carrier frequency of 10,000 Hz required the least amount of mA ($\bar{X}$ = 19.7 mA), the 2,500 Hz carrier frequency was next ($\bar{X}$ = 35.42 mA) and the 5,000 Hz carrier required the highest amount of mA to electrically induce 50% MVC in the subjects studied ($\bar{X}$ = 43.4 mA). Although not statistically significant, there appears to be a trend of decreased mA as the burst mode increases (Figure 14). Overall a mean milliamperage of 32.9 was required. The mA ranged from a maximal mean of 44.1 mA to sufficiently stimulate the quadriceps femoris muscles to 50% MVC at a frequency combination of 50 bursts and a 5,000 Hz carrier

Table 9

ANOVA (Burst VS Carrier Frequency) for EMG Changes (mm^2) Pre VS Post Stimulation

Source	df	SS	F	P
Model	34	409355.1	2.29	0.0002
Error	208	1092272.1	Mean decrease EMG = 58.3 mm^2	
Burst	2	4547.8	0.43	0.6491[A]
Carrier Frequency	2	53365.3	5.08	0.0070
Burst*Carrier Frequency	4	54924.3	2.61	0.0364[b]

[A] = non-significant

[b] = a significant interaction between burst mode and carrier frequency

Figure 13 - Pattern of decreased loss of area under the negative deflection of the EMG curve at various combinations of bursts and carrier frequencies.

MUSCLE ACTION POTENTIAL – AREA UNDER CURVE (mm^2)
PRE vs POST STIMULATION DECREASE
90
80
70
60
50
40
30
20
10
0
50 70 90
2500
50 70 90
5000
50 70 90
10000
BURST / CARRIER FREQUENCY COMBINATION (Hz)
50
70
90

Table 10

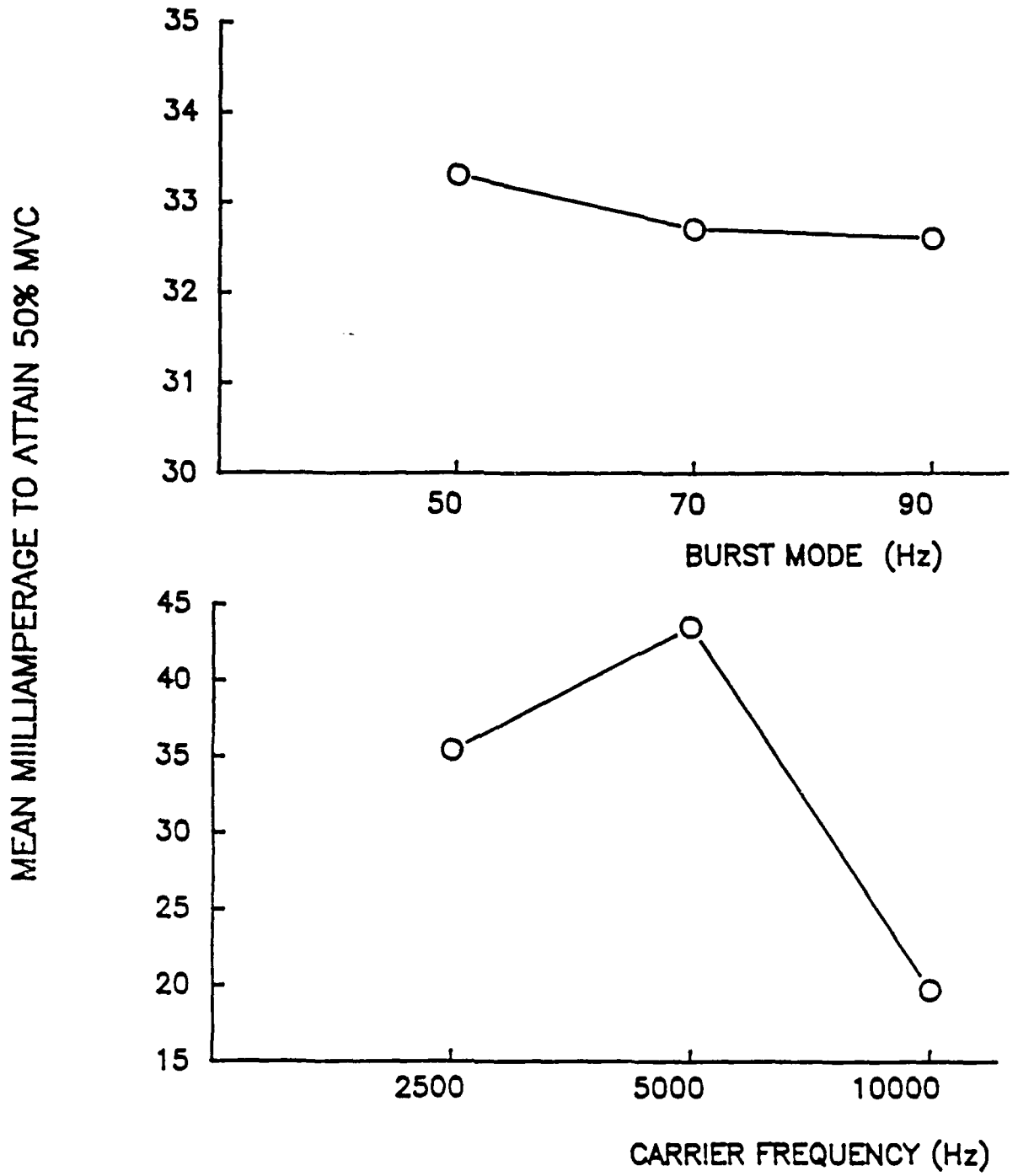
ANOVA (Burst VS Carrier Frequency) for the Mean Milliamperage Necessary to Attain 50% MVC

Source	df	SS	F	P
Model	54	30333.7	22.16	0.0001
Error	208		Mean mA = 32.9	
Burst	2	26.20	0.33	0.7226[A]
Carrier Frequency	2	23646.5	293.62	0.0001[B]
Burst*Carrier Frequency	4	18.13	0.11	0.9780[A]

[A] = non-significant

[B] = A Duncan post hoc test revealed a significant difference among all three carrier frequencies

Figure 14 - Relationship between burst mode (Hz) and carrier frequency (Hz) to the mean milliamperage necessary to attain 50% MVC.


MEAN MIILLIAMPERAGE TO ATTAIN 50% MVC
35
34
33
32
31
30
50
70
90
BURST MODE (Hz)
45
40
35
30
25
20
15
2500
5000
10000
CARRIER FREQUENCY (Hz)

frequency to a low of 19.3 mA required at 90 bursts and a 10,000 Hz carrier frequency (Table 11). The relationship of the combined bursts and carrier frequencies is illustrated in Figure 15.

Subjective Rating of Pain at the Nine Combinations of Bursts and Carrier Frequencies.

An ANOVA discloses that both bursts (50, 70, 90 Hz) and carrier frequencies (2,500, 5,000, 10,000 Hz) significantly influenced the subjective pain rating (Table 12). The subjective pain rating was the only dependent variable investigated in Phase I that was significantly influenced by the burst mode ($\underline{F}$ = 3.12, $\underline{p}$ = .0464). Post hoc analysis of the means shows that a burst mode of 90 Hz was significantly less painful than either the 50 or 70 Hz burst mode. Although the 50 Hz burst mode was deemed the most painful ($\overline{X}$ = 4.86 scale points), it was not significantly different from 70 bursts ($\overline{X}$ = 4.83 scale points). Figure 16 illustrates the trend of decreasing pain rating as the burst mode advances. The carrier frequency also affected the pain rating significantly ($\underline{F}$ = 29.8, $\underline{p}$ = .0001) (Table 12). Post hoc analysis reveals that a 10,000 Hz carrier frequency was significantly more painful ($\overline{X}$ = 5.35 scale points) than either 5,000 Hz ($\overline{X}$ = 4.27 scale points) or 2,500 Hz ($\overline{X}$ = 3.82 scale points). There was no statistical difference between the latter two carrier frequencies. Figure 16 clearly shows an increasing pain rating as the carrier frequency increased. There was a marked variation in the individual pain ratings. The ratings ranged from a low of .2 cm given by two subjects to a high of 9.6 cm also given by two subjects. One of the low pain ratings was given the following stimulation at 90 bursts and a 2,500 Hz carrier frequency, while the other followed a 70 burst, 5,000 Hz

Table 11

Amount of Milliamperage Necessary to Stimulate the Quadriceps Femoris Muscle to 50% MVC

Burst/Carrier Frequency	N	$\bar{X}$	SEM
50/2,500	27	35.7	1.65
50/5,000	27	44.1	2.42
50/10,000	27	20.1	0.95
70/2,500	27	35.6	1.53
70/5,000	27	42.7	1.73
70/10,000	27	19.7	0.40
90/2,500	27	34.9	1.49
90/5,000	27	43.5	1.99
90/10,000	27	19.3	0.42

Figure 15 - Graphic representation of the relationship of the combined bursts and carrier frequencies to the milliamperage necessary to attain 50% MVC.

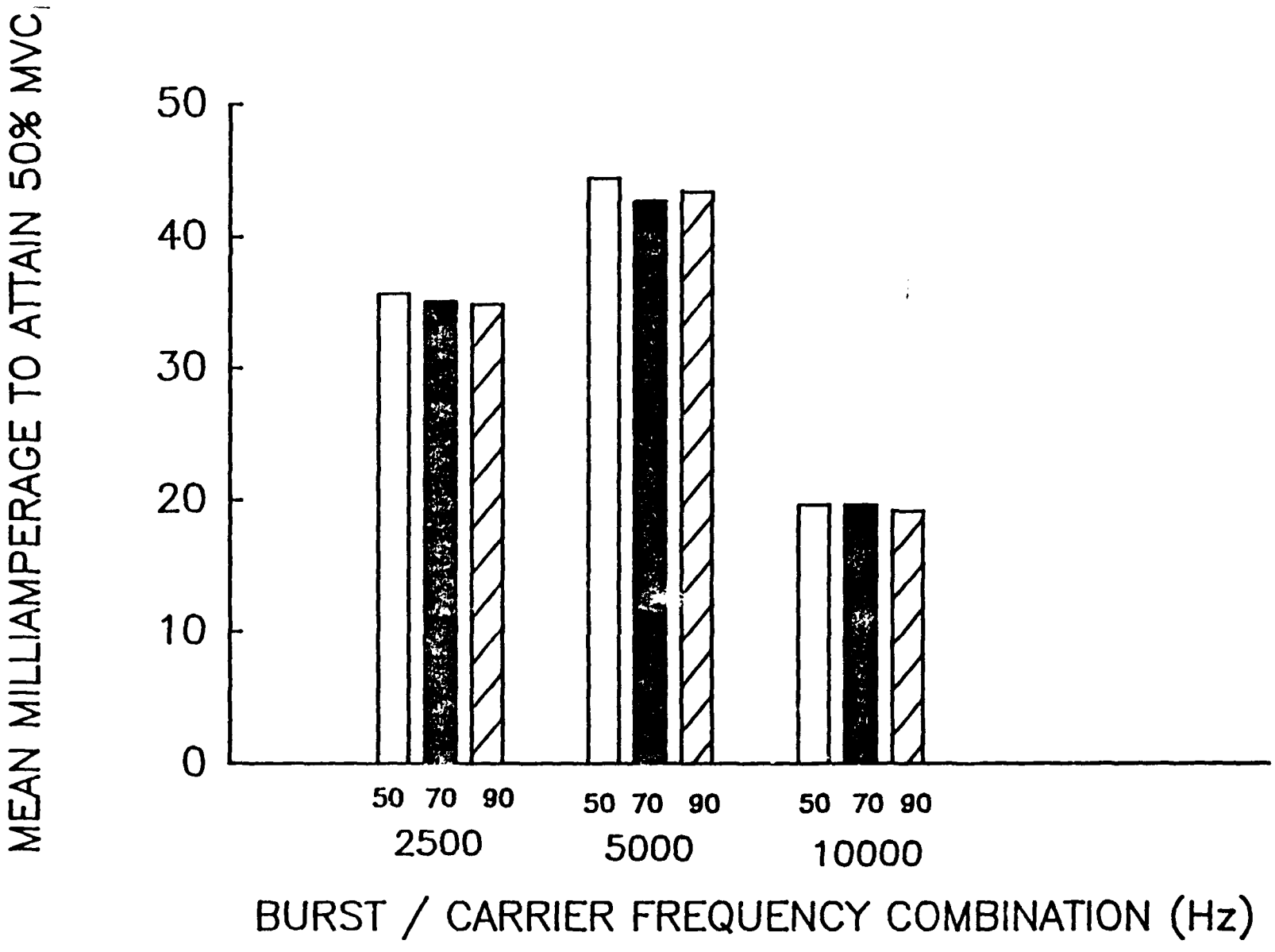

MEAN MILLIAMPERAGE TO ATTAIN 50% MVC
50
40
30
20
10
0
50 70 90
2500
50 70 90
5000
50 70 90
10000
BURST / CARRIER FREQUENCY COMBINATION (Hz)

Table 12

ANOVA (Burst VS Carrier Frequency) for the Pain Ratings of the Nine Combinations of Bursts and Carrier Frequencies

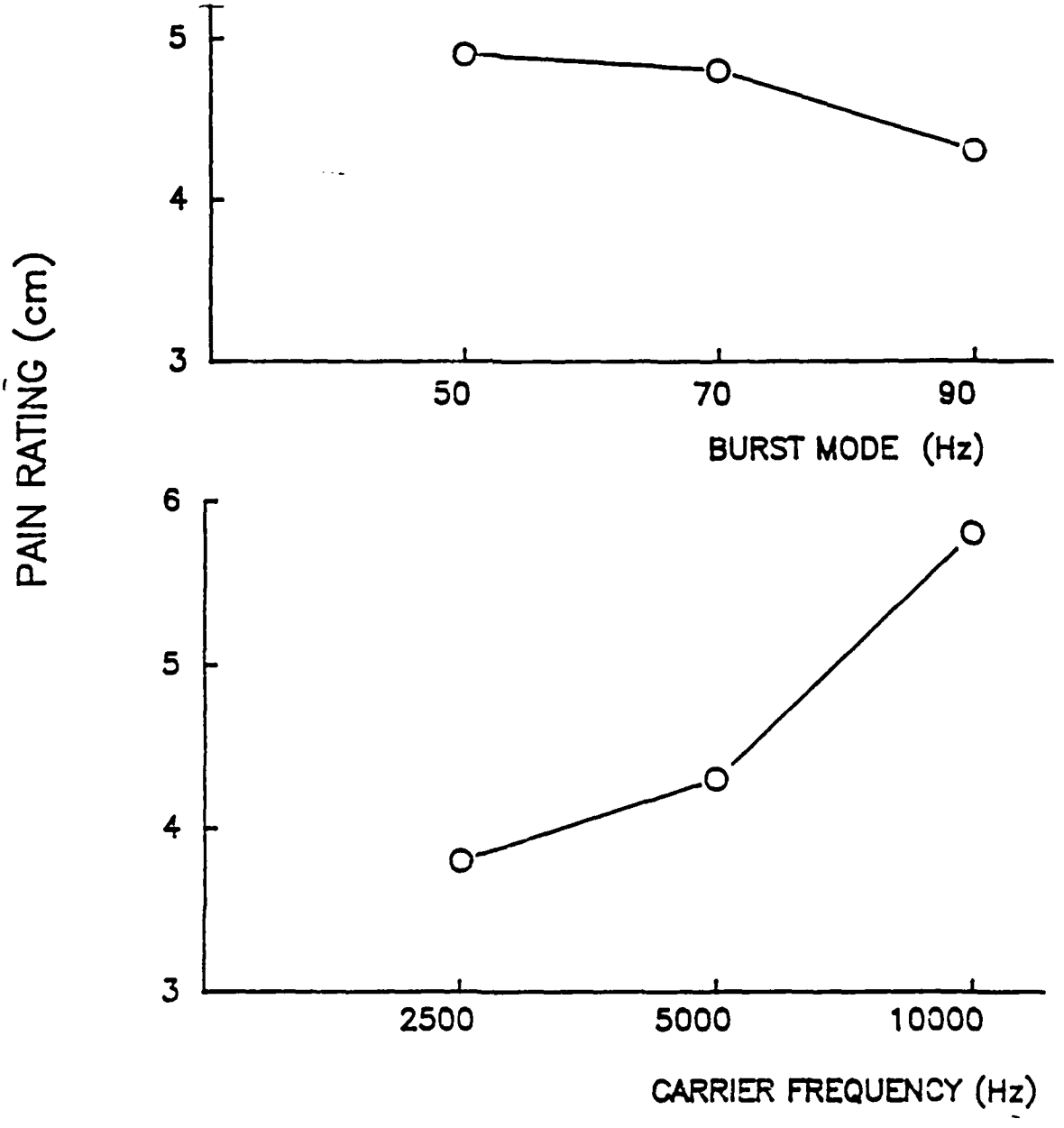
Source	df	SS	F	P
Model	34	741.2	7.11	0.0001
Error	208	638.1	Mean Pain Rating = 4.6 cm	
Burst	2	19.12	3.12	0.0464[A]
Carrier Frequency	2	182.8	29.80	0.0001[B]
Burst*Carrier Frequency	4	9.91	0.81	0.5214[C]

[A] = A Duncan post hoc reveals a significant difference between 90 burst and 50 or 70 bursts. No significant difference noted between 50 and 70 bursts.

[B] = A Duncan post hoc test reveals a significant difference between 10,000 Hz and 2,500 or 5,000 Hz. No significant difference between 2,500 Hz or 5,000 Hz.

[C] = Non-significant interaction

Figure 16 - Depicts trends of decreasing pain rating as the burst mode (Hz) increases and an increasing pain rating as the carrier frequency (Hz) increases.


PAIN RATING (cm)
5
4
3
50
70
90
BURST MODE (Hz)
6
5
4
3
2500
5000
10000
CARRIER FREQUENCY (Hz)

combination. Both of the high ratings involved a 10,000 Hz carrier frequency, one in combination with 50 bursts and the other with the 70 burst mode (Appendix C). Figure 17 shows how subjects rated the combinations of burst and carrier frequency. The 90 burst, 2,500 Hz carrier frequency was rated most favorable with a mean of 3.3 cm (SEM - 0.39) on the VAS, while the 70 burst, 10,000 Hz carrier frequency was the least favorable combination ($\overline{X}$ = 6.3 cm., SEM = 0.40) (Table 13).

One subject was unable to tolerate any of the applied frequency combinations to a level sufficient to produce 50% of her MVC. Although she completed all aspects of this research, the data obtained from her participation were not utilized in the statistical analysis.

Phase II

Phase II utilized the data on pain perception analyzed from Phase I. Of the 27 subjects successfully completing this study, 18 indicated that their preferred frequency combination had a carrier frequency of 2,500 Hz and 9 chose the 5,000 Hz carrier. No subject picked a frequency combination with a 10,000 Hz carrier frequency. The most commonly chosen burst mode was 90 bursts with 13 most favorable ratings, followed by 70 bursts with 8 and 50 bursts with 6 favorable picks (Table 14). The frequency distribution in Table 14 shows that as the number of bursts increased from 50 to 90 Hz, the number of favorable ratings also increased (50 bursts = 6, 70 bursts = 8, 90 bursts = 13). It is interesting to note that this sequential increase is also present when the burst mode is looked at in combination with the chosen carrier frequencies. At 2,500 Hz carrier frequency and 50 bursts there are 4 favorable responses, at 2,500

Figure 17 - Combined effect of burst mode (Hz) and carrier frequency (Hz) on subjective pain rating.

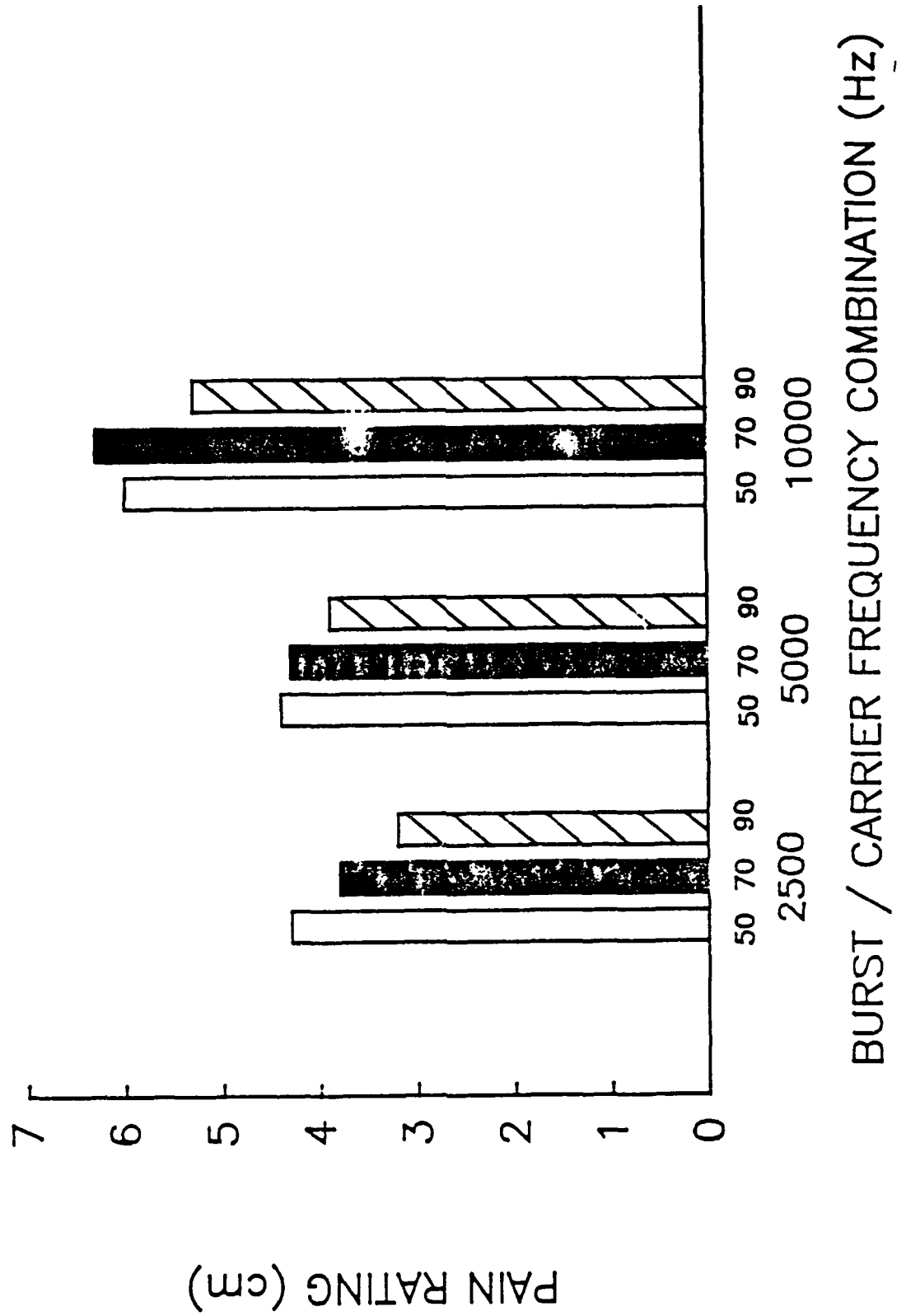

PAIN RATING (cm)
0
1
2
3
4
5
6
7
50 70 90
2500
50 70 90
5000
50 70 90
10000
BURST / CARRIER FREQUENCY COMBINATION (Hz)

Table 13

Pain Rating at the Tested Burst and Carrier Frequency Combinations

Burst/Carrier Frequency	N	$\overline{X}$	SEM
50/2,500	27	4.3	0.36
50/5,000	27	4.4	0.48
50/10,000	27	5.9	0.42
70/2,500	27	3.9	0.43
70/5,000	27	4.3	0.44
70/10,000	27	6.3	0.40
90/2,500	27	3.3	0.39
90/5,000	27	4.2	0.42
90/10,000	27	5.3	0.49

Table 14

Combinations of Bursts and Carrier Frequencies Chosen Most Comfortable

	Carrier Frequency			
Bursts	2,500 Hz	5,000 Hz	10,000 Hz	Total
50	4	2	0	6
70	5	3	0	8
90	9	4	0	13
TOTAL	18	9	0	27

Hz/70 bursts there are 5 choices and at 2,500 Hz/90 bursts there are 9 picks. This sequence is again obvious when looking at the 5,000 Hz carrier frequency with the same pattern of increased favorable responses as the burst mode rate increases (50 bursts = 2, 70 bursts = 3, 90 bursts = 4). Data from Phase I showed that both burst mode and carrier frequency significantly influenced subjects' pain ratings and that overall the 90 bursts, 2,500 Hz carrier frequency was the most comfortable (Figure 17). Figure 18 clearly illustrates that subjects preferred the higher burst modes in the two chosen carrier frequencies.

The Increase in Milliamperage Necessary to Maintain 50 Percent of Each Subject's MVC.

The mean percentage increase in mA necessary to maintain 50% MVC (increments every two stimulations) was 40.5% and ranged from 12 to 90% (Appendix D). Within the two chosen carrier frequencies (2,500 Hz and 5,000 Hz), the percentage increase became greater as the burst rate increased from 50 bursts to 90 bursts (Figure 19). As can be seen in Table 15, the 2,500 Hz carrier frequency tended to require a greater increase in mA ($\overline{X}$ = 40.6%) than a 5,000 Hz carrier frequency ($\overline{X}$ = 33.8%) to maintain 50% MVC.

Analysis of the Difference in Phase I vs. Phase II EMG Changes at Each Subjects Preferred Frequency Combination.

As can be seen in Table 16, in every case tested, the percent decrease in EMG area was greater in Phase II then in Phase I. The mean decrease in EMG areas of the 27 preferred frequencies identified in Phase I was 30.1%. When the same frequency combinations were utilized during Phase II (with the milliamperage being increased every 2 contractions; total = 10) there was a mean decrease of 45.7% in the

Figure 18 - Illustration of the effect on pain rating of the burst mode at the preferred carrier frequencies.

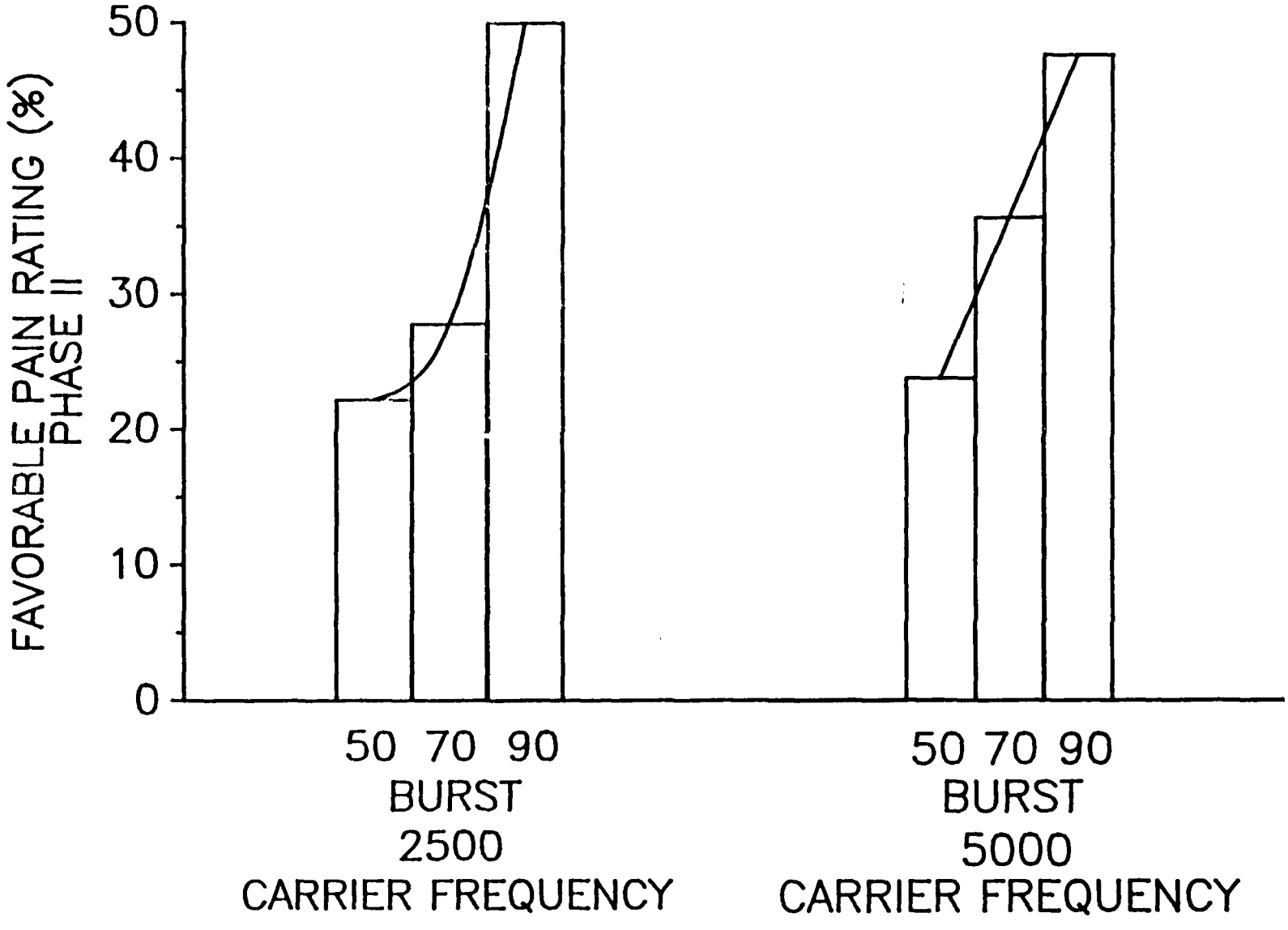

FAVORABLE PAIN RATING (%)
PHASE II
50
40
30
20
10
0
50 70 90
BURST
2500
CARRIER FREQUENCY
50 70 90
BURST
5000
CARRIER FREQUENCY

Figure 19 - Illustration of the percentage increase in milliamperage necessary to maintain 50% MVC at various burst modes (Hz) at the preferred frequencies in Phase II.

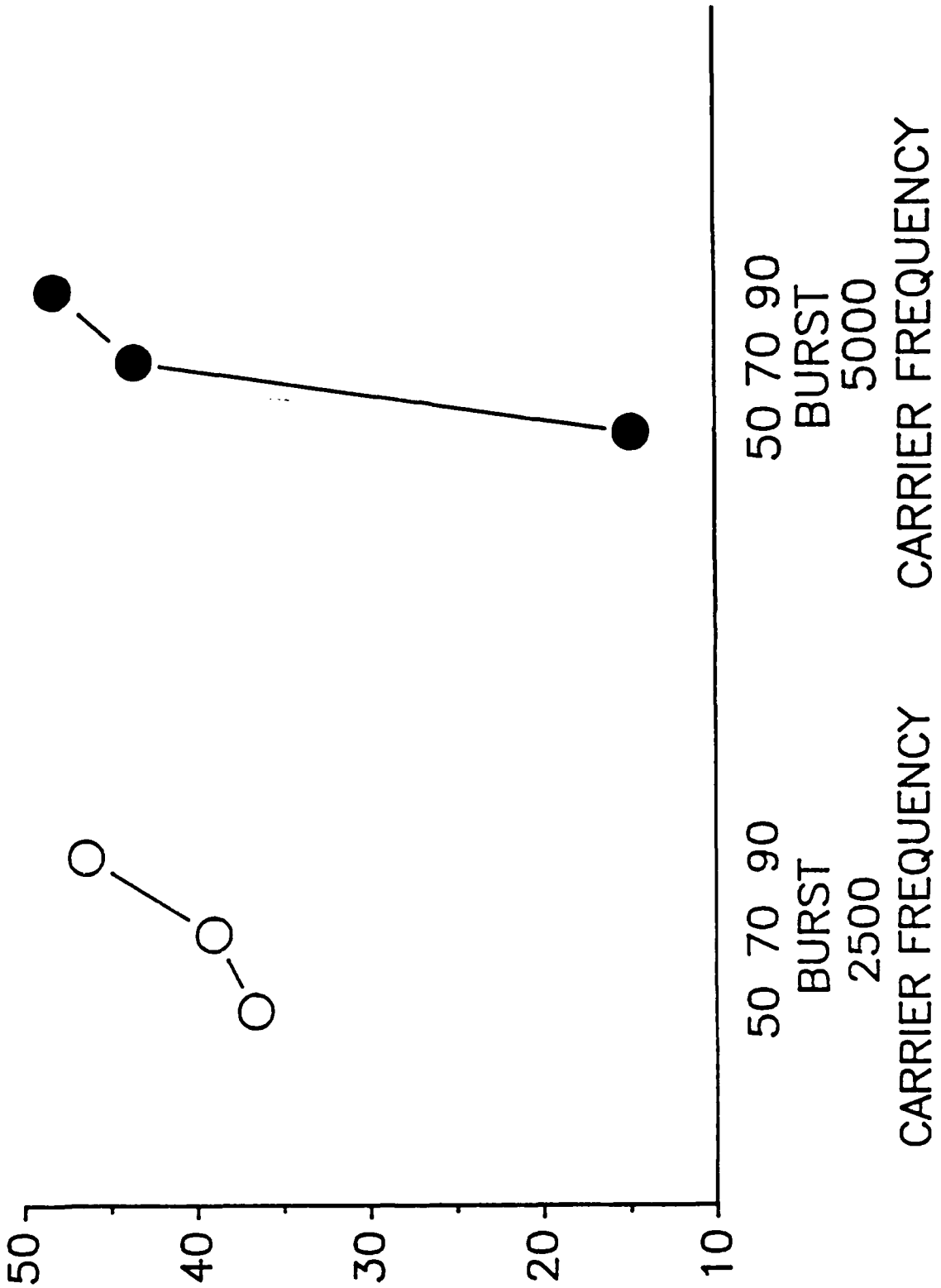

50
40
30
20
10
50 70 90
BURST
2500
CARRIER FREQUENCY
50 70 90
BURST
5000
CARRIER FREQUENCY

Table 15

Percent Increase in Milliamperage Necessary to Maintain 50% MVC in Phase II

	Carrier Frequency	
Bursts	2,500 Hz	5,000 Hz
50	36.3%	14.5%
70	39.0%	41.3%
90	46.4%	45.7%
MEAN	40.6%	33.8%

Table 16

Percentage Change in EMG Area Phase I VS Phase II

Frequency Combination (burst/carrier frequency, Hz)	N	Phase I Decrease in EMG (%)	Phase II Decrease in EMG (%)	Change (%)
50/2,500	4	41.75	47.5	+5.75
50/5,000	2	13.50	49.0	+35.50
70/2,500	5	28.20	45.4	+17.20
70/5,000	3	24.66	36.0	+11.34
90/2,500	9	33.11	47.6	+14.49
90/5,000	4	26.50	45.7	+19.20
MEAN		30.1	45.7	+15.80

areas under the negative deflection of the EMG curve (Table 16). A dependent t-test revealed a significant difference between the percent decrease in EMG area during Phase I stimulation by the preferred frequencies and the decrease in Phase II (t = 1.98, p = .029) (Table 17).

DISCUSSION

Subject Motivation

All of the subjects in this study generated a maximal isometric torque (N.m) of at least 75% of their body weight. This condition of torque and body weight is consistent with the findings of Edwards (1977) who reported similar results.

I feared that some subjects - knowing that they would be stimulated electrically to 50% of their maximal torque reading - would not give a maximal effort and hence, prejudice the results of this study. The correlation between body weight and maximal force in N was also significant (r = .7359, p .0001) (Figure 8). Although Edwards reported a correlation coefficient of r = .91 when comparing body weight to isometric torque, he tested 145 subjects. I believe my subjects were sufficiently motivated and if my sample size was larger than N = 27, the correlation reported in this study would have approached that of Edwards. Maximal volitional effort by each subject was important since the pre- and post-stimulation torque was being quantified and statistically compared. Marsden, et al. (1983) Bigland-Ritchie, et al. (1984) reported that in motivated subjects, all motor units would be utilized during a maximal voluntary effort. The activation of all the available motor units during the required maximal contraction of the quadriceps femoris muscle - recorded before

Table 17

t-Test (Phase I VS Phase II) EMG Changes at Preferred Stimulating Combinations (%)

Source	N	$\overline{X}$	SEM	t	P
EMG Change	27	15.8	7.9	1.98	0.0291

and after the muscle was electrically stimulated - was a necessary criterion for evaluating and comparing the recorded torque and EMG data. The descriptive data and correlation coefficient displayed in this research project convinced me that the subjects tested were expending their maximal effort.

Torque Decrement Following Electrical Stimulation.

A decrease in maximal voluntary isometric torque of 30.3% following electrical stimulation is reported in Phase I of this study. Currier and Mann (1983) reported a 24% decrease in torque following 10 electrically stimulated contractions, while Kots (1976) reported a 20% decrease. The 30.3% decrease found in this study was statistically significant. Further analysis of the data revealed that the burst mode (50, 70, 90 Hz) had no effect on the decrease in torque while the carrier frequency had a significant impact. The data in Figure 9 show that a carrier frequency of 10,000 Hz yields a significantly lower torque decrement ($\overline{X}$ = 26.1 N.m) than either the 2,500 Hz ($\overline{X}$ = 35.1 N.m) or 5,000 Hz ($\overline{X}$ = 34.2 N.m) carrier frequencies. If viewed independently, one might be led to believe that the 10,000 Hz carrier frequency would be the most clinically applicable of the carrier frequencies tested. After all, a carrier frequency that would induce at least 50% of a patient's MVC and affect torque loss less than other clinically utilized carrier frequencies could be adaptable not only to strength enhancing programs (increasing the number of maximal contraction before fatigue) but also to functional electrical stimulators (here maximizing the number of repetitions before fatigue). Solomonow, et al. (1982) and Packman-Braun (1988) cite the high rate of muscular fatigue as the

major drawback to the use of electrical stimulation. However, as will be discussed in a subsequent section, the 10,000 Hz carrier frequency produced an unacceptable level of subject discomfort.

A 24% decrement in torque following ten electrically stimulated muscle contractions of the quadriceps femoris muscle at 60% MVC was contrasted by Currier and Mann (1983) with a 10% decrease following a similar voluntary effort. Cabric (1988) postulates that electrically stimulated muscle contractions show a greater decrease in torque production when compared to voluntary efforts because they have a more pronounced affect on the Type II muscle fibers than on the Type I fibers. The increase in torque decrement may be due to the reported lower threshold to electrical stimulation of the large diameter axons that typically innervate the Type II fibers (Solomonow, et al., 1982). Benton, et al. (1981) confirm this observation and tie the increased rate of fatigue to the preferential stimulation of the Type II fibers during electrical stimulation of skeletal muscles. Komi and Tesch (1979) tied the increased rates of fatigue seen in different muscle groups during electrical stimulation to the proportion of Type II fibers present. Karlsson (1981) also observed that at a given exercise intensity, Type II dominant muscles produce more lactic acid and fatigue more easily than Type I dominant muscles. The decrease in muscle pH associated with increased lactic acid may contribute to peripheral fatigue for a variety of reasons. The most frequently mentioned reasons for muscle fatigue cited in the literature are: inhibition of key glycolytic enzymes (Danforth, 1965; Hermansen, 1981; Ui, 1966); limitation of the interaction of calcium and troponin necessary for actin and myosin to interact (Astrand and Rodahl, 1977;

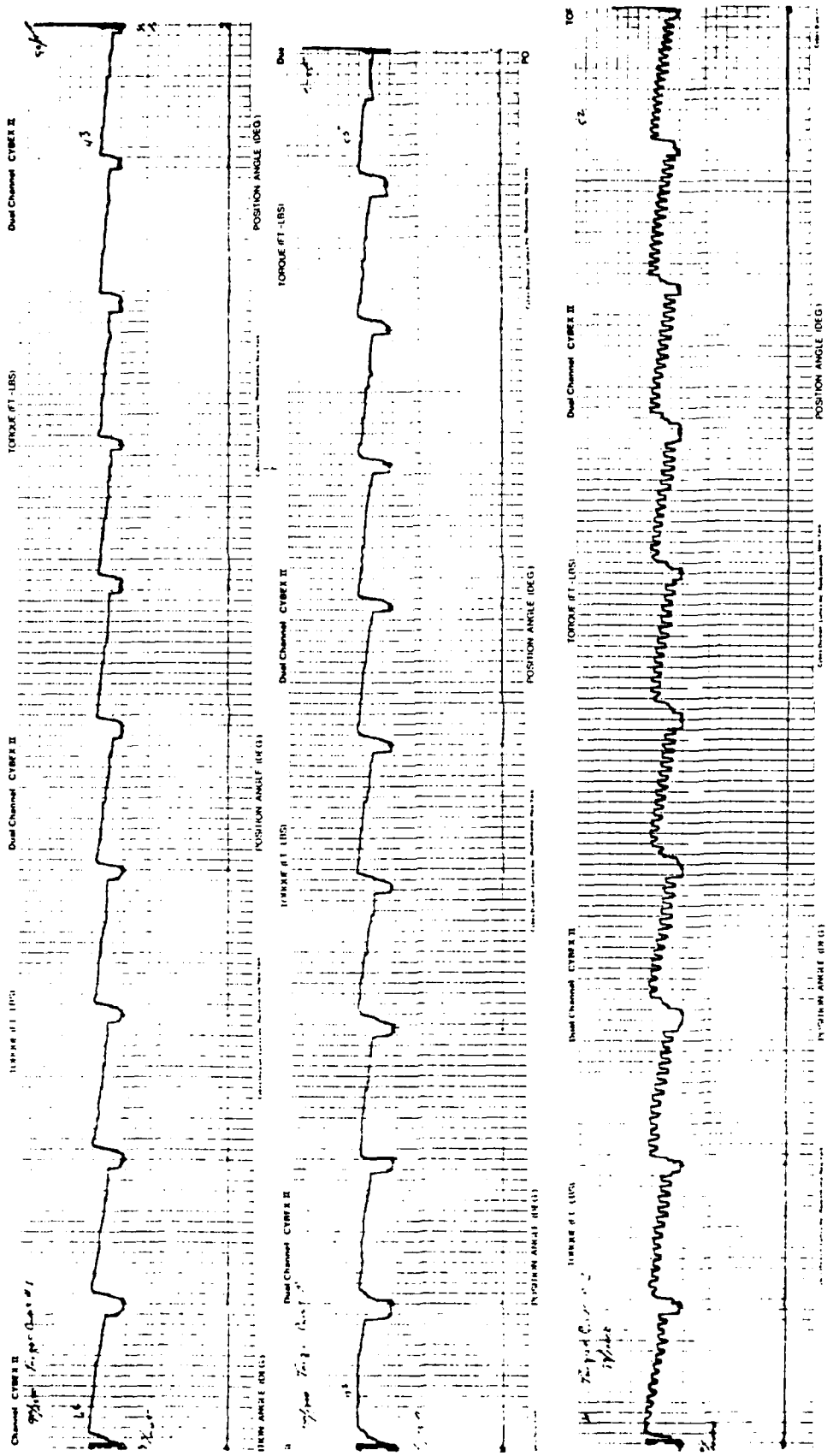
Basmajian and Deluca, 1985; Hermansen, 1981; Robertson and Kerrick, 1979; Tesch, 1980); and the reduction of the conduction velocity of the muscle membrane (Basmajian and DeLuca, 1985; Hagberg, 1981; Petrofsky, 1979; Petrofsky and Lind, 1980). The superficial location of the Type II fibers reported by several researchers may also result in their early activation during electrically stimulated muscle activity (Clamann, 1970; Gollnick, 1983; Johnson, et al., 1973). Appell (1988) refers to a reversal of the "size principle" (Hennamen, 1965) when muscles are electrically stimulated. The larger, more powerful Type II fibers are activated first during the electrically stimulated activity, and since they are more susceptible to fatigue, the increase loss of torque reported is not surprising. There is a preponderance of evidence suggesting that electrical stimulation affects the highly fatigable Type II fibers to a greater extent than the fatigue resistant Type I fibers. This sequence of fiber type activation is the reverse of the natural fiber type recruitment order seen in voluntary contractions. One of the reasons for the high amount of torque decrement reported in this study was the preferential activation of the Type II muscle fibers during the ten electrically induced muscle contractions. No muscle biopsy was obtained in my study and therefore, no definitive conclusions can be drawn concerning muscle pH following electrical stimulation. However, the levels of force decrement reported (30.1%) are much larger than the 10% decrease expected during ten voluntary contractions at nearly the same force levels (Currier and Mann, 1983).

Although during voluntary activity, fatigue at the myoneural junction has been all but ruled out (Bigland-Ritchie, et al., 1986; Bigland-Ritchie, et al., 1983; Marsden, et al., 1983; Merton, 1981) in non-pathologic conditions, fatigue at the myoneural junction may not be the case when fatigue is observed following electrical stimulation of skeletal muscle. Jones, et al. (1979) reported a decreased responsiveness at the myoneural junction when skeletal muscle was stimulated electrically at 80 Hz. Stimulating at 70 Hz, Elmquist and Quastel (1965) noted an increased release of neurotransmitter substances as the rate of electrical stimulation was increased from 30 Hz to 70 Hz. The asynchronous activation of motor units during voluntary efforts, coupled with a natural slowing of motor unit firing frequency precludes this occurrence in normal nerve and muscle. Jones, et al. (1979) reported that fatigue during electrical stimulation could be delayed if the stimulating frequency was gradually lowered from 30 Hz to 20 Hz. The method described by Jones mimics the body's natural slowing of motor unit firing rates during voluntary contractions. The constant rates of muscle stimulation used in the present study may have led to the decreased responsiveness at the myoneural junction previously described by Jones. This decreased responsiveness, along with the preferential activation of the Type II fibers, may have contributed to the decrease in torque reported following electrical stimulation.

Hoskins, et al. (1978) reported that the force decrement during electrical stimulation was related to the stimulating frequency. When stimulating at 30 Hz, Hoskins noted a 10% decrease in muscle torque, but while stimulating at 100 Hz, a 59.6% decrease was reported. The

current study found no statistical difference in the amount of torque loss when the burst modes were compared (Table 5). Perhaps the range (50 Hz, 70 Hz, 90 Hz) was too narrow. Figure 9 shows that as the burst mode increased, the torque loss also increased. This tends to support Hoskins' conclusions. However, it must be emphasized that this trend was not statistically significant (F = 1.20, p = .3042). When carrier frequencies are compared, the amount of torque loss decreases as the frequency rate increases. Decreasing torque loss as the frequency increases is in conflict with Hoskins', et al. (1979) observation as well as the trend noted when burst rates were analyzed in the present study. Perhaps at the narrower pulse duration (PD), the stimulation may be occurring during the absolute refractory period (ARP) of the muscle (10,000 Hz = PD 50 microsecond; 5,000 Hz = PD 100 microsecond; 2,500 Hz = PD 200 microsecond). Virtually every subject tested reported that the 10,000 Hz carrier frequency caused a jerking muscle contraction. This jerking type of contraction can be easily observed when viewing the torque curves in Figure 20. The smooth force decrements of the 2,500 Hz and the 5,000 Hz carrier frequencies can be easily contrasted with the 10,000 Hz curve. The regular pattern of rising and falling torque readings depicted in Figure 20 may indicate that at the 10,000 Hz carrier frequency, the quadriceps femoris muscle is no longer undergoing a smooth tetanic contraction. The jerking contraction may be caused by the short PD of the 10,000 Hz carrier frequency stimulating the muscle during its ARP. Also recall that the 10,000 Hz carrier frequency caused significantly greater subject discomfort than either the 2,500 Hz or 5,000 Hz carrier frequency. This subject discomfort may have caused a contraction

Figure 20 - Typical torque graphs at various burst modes and carrier frequencies. A) 90/2,500; b) 90/5,000; c) 90/10,000.


Dual Channel CYBEX II
TORQUE (FT-LBS)
POSITION ANGLE (DEG)

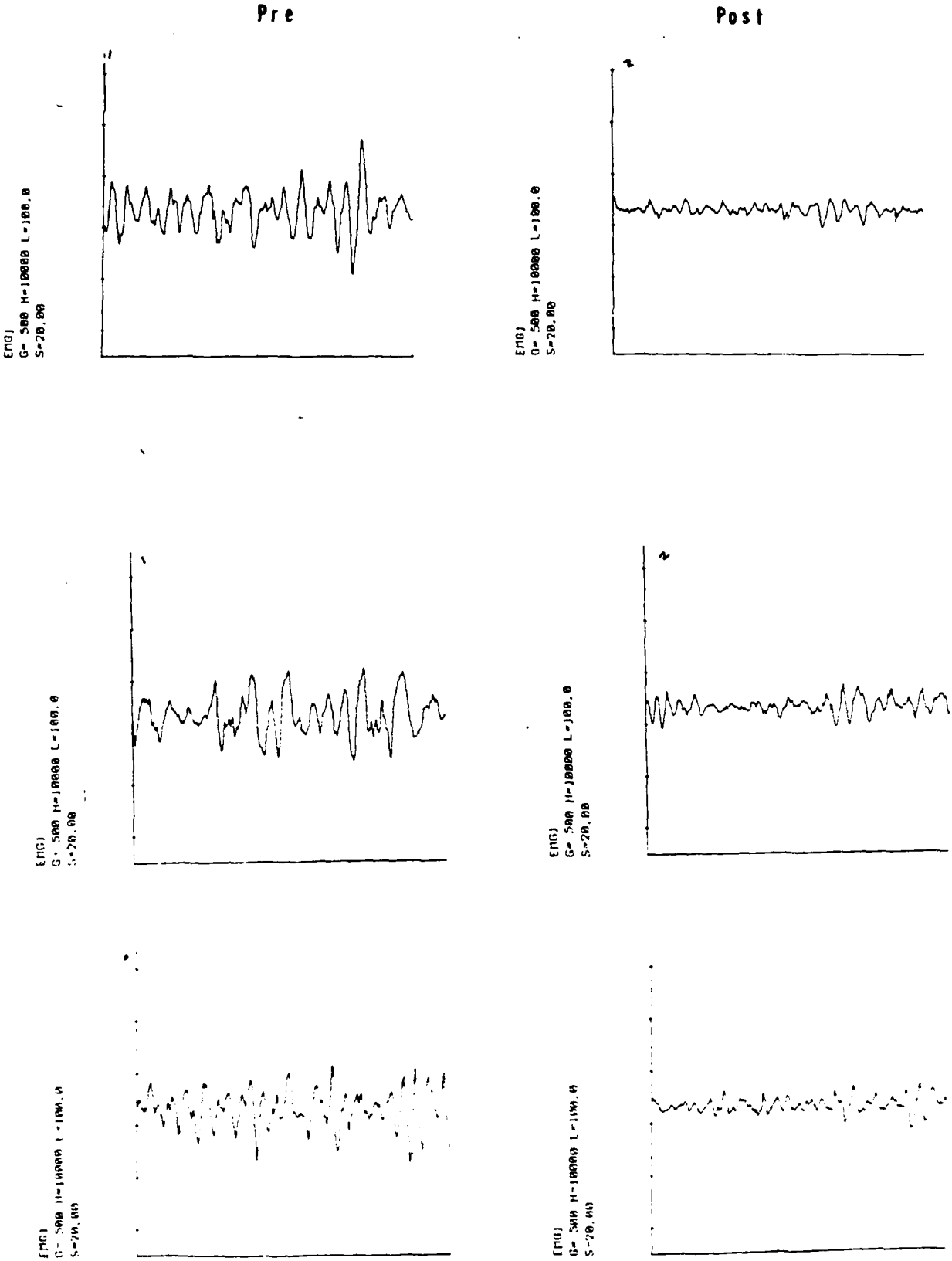
of the hamstring and quadriceps femoris muscles and a distortion in the torque curve. However, the symmetry and consistency of the pattern in Figure 20 leads me to believe that at the 10,000 Hz carrier frequency there was not complete tetany during the 12 seconds of maximal stimulation.

Changes in the Area Under the Negative Deflection of the Muscle Action Potential Following Electrical Stimulation

The nine combinations of burst and carrier frequencies caused a significant decrease in the area under the negative deflection of the muscle action potential (t = 11.54, p = .0001) (Table 6). Recall that Fowler, et al. (1972) reported that the area under the negative deflection of the MAP is indicative of the number of active motor units. Basmajian and DeLuca (1985) concluded that at force levels similar to those tested during this protocol (a minimum of 50% MVC) there is a direct relationship between EMG activity and force. Since there was a significant decrease in EMG activity after electrical stimulation, one can conclude that less motor units were active after stimulation than before electrical stimulation. The decrease in the area under the negative deflection of the EMG curve ranged from a mean of 33.7 mm^2 to 26.3 mm^2. Figure 21 is an example of the pre- and post-stimulation graphs obtained from the Cadwell EMG unit. The reasons for changes in motor unit activity as well as fiber type susceptibility to electrical stimulation was discussed in detail in the previous section.

The ANOVA in Table 9 disclosed an interaction between burst and carrier frequency which necessitated utilization of the graph in Figure 13 to better visualize the effect of the independent variables

Figure 21 - Example of pre- and post-stimulation graphs obtained from the Cadwell EMG Unit. A) 90/2,500; b) 90/5,000; c) 90/10,000


Pre
Post
EMG
G= 500 H=10000 L=100.0
S=20.00

upon the recorded EMG signal. The interaction among the levels of independent variables appears to occur when the 10,000 Hz carrier frequency is employed. Recollect from the previous discussion of torque loss following electrical stimulation that the 10,000 Hz carrier frequency caused significantly lower torque decrements than either the 2,500 Hz or 5,000 Hz carrier frequencies (Figure 11). When viewing the EMG changes, there is a consistent decline in EMG across all the burst modes from carrier frequencies 2,500 Hz to 5,000 Hz. This trend in EMG decrement persists when the 50/10,000 Hz and 70/10,000 Hz combinations are visualized, but the 90/10,000 Hz frequency linking disrupts the trends seen with the other mixes. Basmajian and DeLuca (1985) reported an increase in EMG activity with increased subject anxiety. This increased EMG activity may be an explanation for the unexpected increase in EMG noted at the 10,000 Hz carrier frequency level. The 10,000 Hz carrier frequency was significantly more painful than either the 2,500 Hz or 5,000 Hz combinations. No subject chose a 10,000 Hz carrier frequency combination as the most comfortable. The increased pain experienced during stimulation with the 10,000 Hz carrier frequencies may have increased subjects anxiety to the point of affecting the post stimulation EMG reading. Subject anxiety may partially explain the unexpected increase in EMG activity at the 10,000 Hz carrier frequency. If the 90/10,000 Hz combination is ignored, Figure 13 is quite similar to Figure 11 which documents a decrease in torque loss as the carrier frequency is increased.

Torque and EMG changes following electrical stimulation were quite similar during this experimental protocol. There was an overall 30.3% decrease in torque following electrical stimulation (at any combination) and a 25.3% decrease in EMG area (Tables 4 and 8). However, an insignificant Pearson's product moment of $r = .115$ was drawn between the two variables. This poor correlation between torque and EMG changes may have been due to the unusual interaction of the 10,000 Hz carrier frequency when the area under the negative deflection of the MAP was measured. The increased pain experienced by the subjects when the 10,000 Hz carrier frequency was being used may have contributed to the poor correlation between these variables.

Pain Associated with Electrical Stimulation.

Both the burst mode and the carrier frequency of the stimulating current significantly affected the subjects' pain ratings throughout this study, (Tables 12, 13; Figure 16). The Duncan grouping shows that the 90 Hz burst mode was significantly less painful than either the 50 Hz or 70 Hz burst modes. Although the 50 Hz and 70 Hz burst modes were not significantly different, there is a trend of decreased pain perception among subjects as the burst mode increases (Figure 16). This trend of decreased pain perception is in agreement with Alon's (1987) assertion that as the frequency of stimulation increases, the painful response decreases. Alon notes that as the frequency of a stimulating current increases, it lowers the skin resistance and allows a deeper penetration of the stimulating current than lower frequencies. Since pain conducting nerve fibers are believed to be more superficially located than motor fibers, (DeGirardi, et al., 1984; Alon, 1987), the higher frequency

stimulation is believed to minimize pain fiber stimulation (Alon, 1987). Vodovinik, et al. (1965) and Crochetiere, et al. (1967) also reported a decrease in painful sensory response as the frequency of the stimulation increased. The data in Phase II agree with the Phase I findings as the 90 Hz burst mode was by far the most frequently chosen frequency (Table 14).

In marked contrast to the data on burst modes, the carrier frequency information reported in Phase I and II shows an opposite leaning (Tables 12 and 14, Figure 16). Here, as the rate of the carrier frequency increased, subjects reported more pain. The 10,000 Hz carrier frequency was significantly more painful than that of either 2,500 Hz or 5,000 Hz. The explanation for this reversal of pain/frequency concept may lie in the jerking sensation (i.e., unfused tetanic contraction response) reported by all the subjects tested when this carrier frequency was used. As mentioned previously, the jerking muscle contractions caused by the 10,000 Hz carrier frequency may have caused increased subject apprehension. Several subjects stated that "it felt like my muscles were tearing." The torque curves in Figure 20 depicts the periodic contraction and relaxation observed during 10,000 Hz stimulation. This apprehension caused several subjects to contract their hamstring muscles in an attempt to alleviate the jerking motion. I cautioned the subjects to avoid any hamstring activity. I was able to control this tendency by visual inspection of the hamstring muscles and in one case, by palpation of the hamstring muscle group. Because of the subject discomfort associated with the 10,000 Hz carrier frequency, I believe that the 10,000 Hz carrier frequency mode is unacceptable as a clinical tool. As stated

previously, no subject found any burst mode accompanied by a 10,000 Hz carrier frequency to be an acceptable combination. The range of discomfort reported in this study was from .2 cm to 9.6 cm (Appendix C). Both the 9.6 cm ratings involved a 10,000 Hz carrier frequency. When the bar chart depicted in Figure 17 is viewed, the combination of 90 bursts and 2,500 carrier frequency received the most favorable rating during Phase I. Even though this finding of higher carrier frequencies causing increased pain is strongly supported by the data, I feel the high pain ratings were partially due to the jerking contractions of the quadriceps femoris muscles during stimulation. These jerking contractions were especially obvious when a 10,000 Hz carrier frequency torque curves are compared to the 2,500 Hz and 5,000 Hz torque curves (Figure 20).

Although subjects rated the discomfort immediately following each stimulation sequence, an interesting pattern of delayed onset of muscle soreness (DOMS) emerged as the study progressed. Approximately 50% of the subjects report delayed onset of muscle soreness 24 to 48 hours following at least one stimulation session. Two subjects reported severe soreness 24 hours after stimulation and described the pain as similar to what they experienced after resuming their weight lifting regime after a long layoff. McCully and Faulkner (1985) demonstrated that DOMS is associated with eccentric exercise and is not demonstrated following isometric exercise. Garrett (1985) describes the DOMS following sudden high intensity muscle contractions, while Friden, et al. (1981) report that the majority of the muscle fiber damage associated with delayed muscle soreness was to the Type II fibers. The association of delayed muscle soreness to

sudden high intensity muscle contractions and to Type II fiber damage lead me to believe that following electrical stimulation, especially at 10,000 Hz carrier frequency, there is an increased risk of delayed muscle soreness.

The Changes in Milliamperage Necessary to Produce 50 Percent of a Subject's MVC at Various Combinations of Bursts and Carrier Frequencies in Phase I and II.

The ANOVA in Table 10 reveals that only the carrier frequencies of the tested stimulation combinations significantly influenced the mA necessary to produce 50% of a subject's MVC in Phase I. The Duncan post hoc analysis shows no statistically significant difference between burst modes, but Figure 14 reveals a trend of decreased amounts of milliamperage necessary to produce 50% of a subject's MVC as the burst frequency increased from 50 to 90 Hz. On the other hand, Figure 14 shows that 10,000 Hz carrier frequency required significantly less milliamperage to stimulate the quadriceps femoris muscle to the required level ($\overline{X}$ = 19.7 mA). A 2,500 Hz carrier required significantly more mA than 10,000 Hz ($\overline{X}$ = 35.4 mA), but significantly less than 5,000 Hz ($\overline{X}$ = 43.4 mA) which required the most milliamperage to reach the goal of 50% MVC. Figure 15 illustrates that when bursts and carrier frequencies are combined, a combination of 90 Hz burst and 10,000 Hz carrier frequency is the most efficient pairing ($\overline{X}$ = 19.3 mA, SEM = 0.42 mA) while the 50 Hz burst, 5,000 Hz carrier frequency combination was the least effective ($\overline{X}$ = 44.1 mA, SEM = 2.42 mA). The reason for these differences in milliamperage is not clear when examining the Phase I data. Recall from the discussion of the pain data that the 10,000 Hz carrier frequency appears to be

unacceptable for clinical use. When the frequency combinations containing 10,000 Hz are eliminated, the 90 Hz burst, 2,500 Hz carrier frequency emerges as the combination that yields 50% MVC with the least amount of milliamperage (Figure 17).

When the milliamperage is periodically increased in Phase II to maintain the desired 50% MVC, the 90 Hz burst, 2,500 Hz carrier frequency required the greatest percentage increase (Table 14). The fact that 90 Hz burst and 2,500 Hz carrier frequency required the largest increase is not surprising as in Phase I a burst mode of 90 Hz caused the greatest decrease in torque, while a carrier frequency of 2,500 Hz produced similar results (Figure 9). The increased amount of milliamperage necessary to maintain 50% MVC when the 90 Hz burst, 2,500 Hz carrier frequency was used is because of the increased torque loss caused by this combination. The more fatiguing the burst/frequency combination, the greater the increase in milliamperage necessary to maintain a specified torque reading. At each subject's chosen frequency from Phase I, no subject was unable to tolerate the increase in milliamperage needed to produce a muscle contraction equivalent to 50% MVC. One subject sustained a 90% increase in milliamperage to maintain 50% of his MVC without complaining of increased pain. Once a comfortable burst/carrier frequency combination is identified by a subject, he can better tolerate increases in stimulating intensities than when the optimal burst/carrier frequency is not applied. The key to the clinical use of this modality appears to lie in the identification of a frequency combination acceptable to the subject undergoing high intensity electrical stimulation.

Chapter 5

SUMMARY, CONCLUSION, AND RECOMMENDATIONS FOR FURTHER STUDY

Summary

The purpose of this study was to investigate what effect electrically stimulating human skeletal muscle at various combinations of burst modes and carrier frequencies has on muscle fatigue and pain perception. By using human subjects and clinically available stimulating combinations, this investigator hopes that the conclusions drawn from this study will enhance the use of this modality in physical therapy and sports medicine clinics.

During Phase I of this study, I found that electrical stimulation of the quadriceps femoris muscle by each of the nine combinations of bursts and carrier frequencies brought about 50% MVC in each subject tested. All the tested frequency combinations significantly reduced the torque following the tenth muscle contraction recorded by the Cybex II dynamometer ($\overline{X}$ = -30.3%). Further differentiation of the data showed that the carrier frequency of the stimulating current had a significant impact on decreasing torque, while the burst mode of the current did not affect the change in torque. The Duncan post hoc analysis of the data revealed that the 10,000 Hz carrier frequency caused significantly less torque decrement than either the 2,500 Hz or 5,000 Hz carrier frequencies. The latter two carrier frequencies were not significantly different from each other. However, there was a trend showing that as the rate of the burst mode increased from 50 to 70 to 90 Hz, a greater

amount of torque loss was observed. The 70 Hz burst mode coupled with the 2,500 Hz carrier frequency caused the greatest torque loss while the 50 Hz burst/10,000 Hz carrier frequency caused the least torque decrement following the tenth muscle contraction.

There was a statistically significant decrease in the EMG (negative deflection of the MAP) recorded pre- and post-stimulation of the quadriceps femoris muscle ($\overline{X}$ = -25.3%). As in the torque data, only the carrier frequency significantly affected this measurement and a trend of a greater decrease in EMG area was noted as the burst mode increased. Post hoc testing of the carrier frequency demonstrated that the 2,500 Hz carrier frequency caused a significantly greater loss of EMG area than 10,000 Hz or 5,000 Hz carriers. The 10,000 Hz and 5,000 Hz carrier frequencies were not significantly different from each other.

In Phase II only the subject's most tolerable frequency combination was utilized. When the decrease in EMG recordings from Phase I was compared to the decreases of the same frequency combinations in Phase II, a significantly greater loss of EMG area under the negative deflection of the MAP was realized in Phase II.

Although both torque and EMG readings decreased significantly following a bout of 10 electrically induced muscle contractions, there was a non-significant correlation between these two variables. In contrast to the preceding two dependent variables, both burst mode and carrier frequency significantly influenced the pain ratings of the subjects tested. The 90 Hz burst mode was significantly less painful than either the 50 Hz or 70 Hz current configuration. The 50 Hz and 70 Hz burst modes were not significantly different from each other. A trend of increased subject tolerance was observed as the burst mode increased.

Interestingly, the carrier frequency data showed a trend contradictory to the burst mode results. As the carrier frequency increased, the subjects' rating of perceived pain became elevated. When Phase I pain data were analyzed, the combination of 90 Hz burst mode and 2,500 Hz carrier frequency was preferred by 67% of the subjects tested. This burst/carrier frequency combination supports the trends noted when Phase I data were examined. No subject chose a frequency combination that contained a 10,000 Hz carrier frequency.

When all the tested frequency combinations ($N = 9$) from Phase I were grouped, a mean intensity of 32.8 mA was required to provoke the required 50% MVC. When viewed individually, the 90 Hz burst and 10,000 Hz carrier frequency required the lowest intensity, while the 50 Hz/5,000 Hz burst/carrier frequency required the greatest intensity. When the independent variables were viewed individually, only the carrier frequency exhibited a significant influence on the mA necessary to bring about 50% MVC. The 10,000 Hz carrier required the least milliamperage of current, while the 5,000 Hz carrier required the most to achieve 50% MVC.

During Phase II - when the milliamperage was periodically increased after each two contractions - the 90 Hz burst mode coupled with the 2,500 Hz carrier frequency required the greatest increase in milliamperage to maintain the 50% MVC torque reading. Recall that during Phase I, the higher burst modes and the lower carrier frequencies tended to cause the greatest loss of torque (Figure 10). Phase II confirms this finding. One would expect a greater increase in mA to be required to maintain 50% MVC as the stimulating current becomes more fatiguing.

Conclusion

Considering the limitations of this study, the findings justify the following conclusions:

1. Electrical stimulation of human skeletal muscle at an amplitude sufficient to produce 50% of a healthy subject's MVC results in a decrease in torque and a decrease in the area under the negative deflection of the EMG signal recorded from the stimulated muscle. The degree of torque loss or EMG area decrease is dependent upon the frequency and intensity of the stimulation current.

2. The amplitude of stimulation (mA) necessary to bring about 50% MVC of a given muscle group is dependent upon the frequency of the stimulating current. All of the frequency combinations used in this study were capable of stimulating the quadriceps femoris muscle to produce 50% MVC.

3. Subjects' pain tolerance is contingent upon the stimulating frequency and the intensity of stimulation. Variations in either the burst mode or carrier frequency of a stimulating current can alter a subject's pain response.

4. When torque production decreases as a result of electrical stimulation, an increase in current intensity will re-establish the desired torque level (50% MVC).

5. In the context of this study, there was no correlation between torque and EMG changes following electrically induced muscle stimulations.

6. In addition to the discomfort encountered during electrical stimulation of the quadriceps femoris muscle to produce 50% MVC, there may be delayed onset of muscle soreness occurring 24 to 48 hours after stimulation.

Recommendations for Further Study

The use of muscle biopsies both pre- and post-stimulation would have allowed an objective review of the enzyme changes occurring after electrically induced muscle contractions in humans and make additional assumptions as to what is altering the enzyme composition within human muscles. This study of muscle enzymes would have entailed a muscle biopsy and required additional review by the Human Investigations Review Committee.

I was only able to speculate on where the fatigue (decreased torque production) was occurring in the chain of motor activity beginning at the motor cortex and terminating with the interaction of actin and myosin. Further analysis of the "M" wave both pre- and post-stimulation would have allowed me to objectively view changes in the motor unit activity. Again, as with the addition of a muscle biopsy, this enzyme study procedure entails an increase in subject discomfort.

Allowing subjects to maximally contract their quadriceps femoris muscle while being electrically stimulated may have yielded additional information on fatigue and pain perception. Clinically, when patients are allowed to maximally contract the muscle being stimulated, far greater amplitudes of stimulation are tolerated.

APPENDIX A

Subject Consent Form

CONSENT FORM

TITLE OF STUDY

Effect of Variation in the Burst Mode and Carrier Frequency of High Intensity Electrical Stimulation on Muscle Fatigue and Pain Perception in Healthy Subjects.

I, , consent to participate in a research study under the direction of James G. Rooney, P.T. and Dean P. Currier, P.T., Ph.D., to be conducted at the University of Kentucky.

I understand that the purpose of the study is to determine the effect that variations in the burst mode and carrier frequency have on muscle fatigue and pain perception. The carrier frequency (the number of cycles per second) will be interrupted periodically (burst mode) and the various combinations of burst mode and carrier frequency statistically analyzed to determine the effect of various combinations on fatigue and pain.

A thorough description of the procedures to be used have been explained to me and I understand that there may be discomfort in the thigh muscles during stimulation. The discomfort can best be described as a dull cramping sensation in the thigh muscle. I also understand that I will be able to control the intensity of the stimulator during the experimental session. The procedure is as follows: 1) during a familiarization session, my height, sex and weight will be recorded.

2) My maximum isometric knee extensor torque will be determined using the Cybex isokinetic exercise unit. The highest reading of these repetitions will be recorded. 3) I will be given time to familiarize myself with the Electrostim 180-2i high intensity electrical stimulation unit. 4) Following the familiarization session, there will be three additional experimental sessions. During each of these experimental sessions, I will randomly receive one of three predetermined burst modes (50, 70, 90 Hz) combined with a randomly chosen carrier frequency (2,500, 5,000, 10,000 Hz). I will receive ten stimulations with each combination and experience three combinations per session. 5) Immediately following the tenth stimulation, I will be asked to rate the discomfort experienced, using a visual scale. 6) A final experimental session will determine the effect of incrementing the intensity of stimulation on fatigue, pain and torque. The least painful stimulating combination from the first three sessions will be used and I will control the intensity. Again, ten stimulations will be given.

I understand that it will be necessary for me to receive electrical stimulation during four to five experimental sessions at various frequencies and intensities of stimulation. Each session will last forty-five minutes.

I understand that I may withdraw my consent and discontinue participation in the research at anytime without prejudice to me.

I understand that, in the event of physical injury resulting from the research procedure in which I am to participate, no forms of compensation are available. Medical treatment may be provided at my own expense; by the Veterans Administration Medical Center, if I am eligible; or at the expense of my health care insurer (i.e., Medicare, Medicaid,

BC/BS, etc.) which may or may not provide coverage. If I have questions, I should contact my insurer.

I have been informed of the various contraindications and risks which would keep me from participating in this study. These include cardiovascular problems and pacemakers.

I authorize James G. Rooney and Dean P. Currier to keep, reserve, use and dispose of the findings from this research with the provision that my name not be associated with any of the results.

I have been given the opportunity to ask, and have answered, any questions concerning the procedures to be used during this research. I understand that I will be paid twenty-five dollars by check approximately six weeks after completion of the study pending approval of a grant from the Kentucky Chapter of the American Physical Therapy Association.

Questions have been answered to my satisfaction. I have read and understand the contents of this form and have received a copy.

Witness	Date
Participant	Date

I have explained and defined in detail the research procedure in which the subject has consented to participate.

Signature	Date

APPENDIX B

Body Weight/Height/Torque Data

Subject	Body Weight (kg)	Height (cm)	Torque (N.m)	Force (N)
1	72.7	183	219.6	726
2	72.7	165	219.6	726
3	77.2	183	223.7	739
4	66.0	173	146.4	484
5	70.4	180	204.7	677
6	68.1	178	196.6	650
7	80.9	180	223.7	740
8	79.5	183	199.3	659
9	81.8	183	220.9	730
10	63.6	175	172.2	569
11	77.2	175	196.6	650
12	93.1	193	230.5	762
13	79.5	183	220.9	731
14	73.1	180	197.9	654
15	54.5	163	146.4	484
16	76.3	183	244.0	807
17	61.3	173	196.6	650
18	82.7	183	204.7	677
19	77.2	180	219.6	726
20	65.0	175	195.2	645
21	67.2	175	196.6	650
22	54.5	160	146.4	484
23	81.8	168	204.7	677
24	60.9	175	199.3	659
25	58.8	165	196.6	650
26	61.3	175	149.1	493
27	73.6	178	196.6	650

APPENDIX C

Pain Rating/Burst Mode - Frequency Combination

Pain Rating

Burst Mode/Carrier Frequency

Subject	50/2500	70/2500	90/2500	50/5000	70/5000	90/5000	50/10000	70/10000	90/10000
1	3.7	.9	2.3	1.5	.2	1.1	3.6	8.2	2.6
2	3.7	8.2	4.9	7.0	8.4	6.6	4.6	4.7	6.7
3	2.7	2.7	1.4	2.9	2.8	1.7	5.8	6.2	6.0
4	4.4	4.6	2.5	2.7	1.8	3.7	6.5	6.2	2.4
5	2.6	3.7	6.2	5.2	5.4	4.0	5.3	7.6	9.5
6	5.9	3.8	3.4	7.8	4.2	5.3	7.5	7.9	5.8
7	5.1	7.7	.3	2.4	4.1	4.3	.9	1.6	5.7
8	6.3	5.5	5.1	6.3	6.5	6.2	6.7	5.5+	6.7
9	7.5	5.7	2.3	8.7	.6	1.7	9.2	8.6	1.5
10	5.7	8.0	7.0	8.5	8.9	5.8	9.6	9.2	9.2
11	4.8	3.7	4.4	4.5	4.0	6.0	6.7	8.2	7.4
12	4.7	5.3	1.7	2.0	5.4	3.8	7.1	7.8	8.1
13	6.7	2.7	.9	1.3	6.2	1.7	6.3	5.0	3.1
14	3.7	1.1	.4	3.0	1.8	.3	4.5	6.7	2.0
15	5.6	4.3	4.7	5.9	5.7	4.2	6.1	6.9	9.4
16	2.2	5.8	5.2	5.9	4.5	7.5	6.8	2.2+	2.3
17	4.8	3.9	2.7	.4	2.7	1.7	6.3	5.3	3.1
18	2.3	.5	1.8	1.5	3.8	.9	3.2	6.9	3.4
19	2.4	2.3	2.0	4.6	5.0	2.7	7.5	4.1	7.0
20	5.7	7.0	5.7	6.8	7.7	8.8	9.3	9.6	9.2
21	3.6	3.1+	4.1	3.2	5.2	3.1	6.2	5.3	4.6
22	.8	.8	.2	3.2	2.1	4.7	.3	4.1	3.5
23	1.2	2.3	.5	6.3	1.1	4.2	4.6	7.6	.7
24	7.5	2.1	5.1	8.1	3.6	2.9	7.1	9.1	4.4
25	1.6	2.3	4.4	1.1	5.0	6.9	5.5	3.3	5.6
26	5.3	4.3+	5.2	5.6	6.2	4.3	7.1	7.5	7.0
27	4.6	1.9	2.7	2.0	2.2	2.4	7.0	6.1	5.7

APPENDIX D

Milliamperage Needed to Attain 50% MVC

MILLIAMPERAGE NEEDED TO ATTAIN 50% MVC

Burst Mode/Carrier Frequency

Subject	50/2500	70/2500	90/2500	50/5000	70/5000	90/5000	50/10000	70/10000	90/10000
1	30	30	35	39	38	40	17	20	18
2	32	41	52	52	56	50	22	22	22
3	40	34	48	41	36	50	20	20	21
4	50	36	41	46	58	45	22	20	20
5	22	26	22	30	32	30	13	16	17
6	38	32	36	61	52	38	20	21	21
7	50	56	35	60	51	71	19	19	19
8	31	30	28	33	35	33	28	17	15
9	42	38	30	58	34	41	24	22	14
10	24	37	40	50	45	38	22	22	24
11	36	41	36	38	46	36	20	21	20
12	30	35	28	38	39	40	15	18	17
13	42	40	40	40	40	42	26	21	20
14	37	36	38	38	32	40	19	20	21
15	30	24	23	32	41	28	13	14	20
16	42	50	40	60	42	61	22	23	22
17	38	48	32	40	44	41	20	18	18
18	24	22	22	31	32	32	12	10	18
19	41	40	28	54	58	50	18	18	20
20	30	25	27	42	30	30	21	20	18
21	32	32	38	48	36	50	25	20	18
22	30	30	32	41	40	50	19	21	14
23	60	40	48	80	62	50	17	22	19
24	32	30	32	36	42	42	18	19	20
25	31	34	42	32	40	56	20	17	20
26	31	30	32	30	47	33	19	20	20
27	40	41	38	52	50	57	21	21	22

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